APRIL, 1927

International Abstract of Surgery

Supplementary to

Surgery, Gynecology and Obstetrics

EDITORS

FRANKLIN H. MARTIN, Chicago
SIR BERKELEY MOYNIHAN, K.C.M.G., C.B., Leeds
PAUL LECÈNE, Paris

SUMNER L. KOCH, Abstract Editor

DEPARTMENT EDITORS

DEAN D. LEWIS, General Surgery
CHARLES B. REED, Gynecology and Obstetrics
LOUIS E. SCHMIDT, Genito-Urinary Surgery
PHILIP LEWIN, Orthopedic Surgery
ADOLPH HARTUNG, Roentgenology
JAMES P. FITZGERALD, Surgery of the Eye
FRANK J. NOVAK, Jr., Surgery of the Ear, Nose and Throat
CARL A. HEDBLOM, Chest Surgery

CONTENTS

I. Index of Abstracts of Current Literature iii
II. Authors ix
III. Editor's Comment x
IV. Abstracts of Current Literature 259-332
V. Bibliography of Current Literature 333-360

Editorial communications should be sent to Franklin H. Martin, Editor, 54 East Erie St., Chicago
Editorial and Business Offices: 54 East Erie St., Chicago, Illinois, U. S. A.
Publishers for Great Britain: Bailliere, Tindall & Cox, 8 Henrietta St., Covent Garden, London, W.C.

CONTENTS—APRIL, 1927

ABSTRACTS OF CURRENT LITERATURE

SURGERY OF THE HEAD AND NECK

Head

DIXON, O. J.: The Pathological Examination in Cavernous Sinus Thrombosis as a Guide to the Diagnosis, Prognosis, and Treatment 259

STAMMERS, F. A. R.: Pre-Auricular Fistulæ 259

GROVE, L. W.: Mixed Tumors of the Parotid Gland and Their Removal, with a Consideration of the Seventh Nerve 259

Eye

SCHILD, E. H.: An Unlearnable Prism Test for Suspected Malingering 260

IRONS, E. E., and BROWN, E. V. L.: Recurrence of Iritis as Influenced by the Removal of Infections: Summary of Fifty Cases 260

ZENTMAYER, W.: The Prostate as a Remote Focus of Infection in Ocular Inflammations 260

MILLS, L.: Ocular Disease Occurring in the Course of Non-Dysenteric Amœbiasis 260

Ear

GOLDSTEIN, M. A.: The Relation of Tactile Impression and Hearing Perception 261

TAIT, J.: Ablation Experiments on the Labyrinth of Frogs 261

BIEHL, C.: Perforation of the Fenestra Rotunda for Therapeutic Purposes 262

LEIDLER, R.: The Indications for Opening the Labyrinth 262

LILLIE, H. I., and STARK, W. B.: The Insidious, Symptomless, Destructive Effect of Cholesteatome 263

Nose and Sinuses

WILLCOX, SIR W.: Nasal Sinusitis as a Cause of Toxæmia 263

MCMAHON, B. J.: The Pathology of Spheno-Ethmoidal Sinusitis 264

Mouth

BROWN, G. V. I.: The Surgical Treatment of Cleft Palate 264

THOMPSON, J. E.: A Septal Flap in the Closure of Unilateral Clefts of the Palate 264

FIGI, F. A.: Actinomycosis of the Tongue: Report of Twelve Cases 265

Pharynx

NEW, G. B. and DECKER, W. J.: Pharyngeal Sinus with Cervical Pott's Disease; Report of Six Cases 265

Neck

HUDSON, R. V.: The So-Called Branchiogenetic Carcinoma: Its Occupational Incidence and Origin 265

MENNE, F. B., JOYCE, T. M., and VON HUNGEN, A. P.: Thyroid Disturbances: A Clinicopathological Study of 300 Instances 266

KEITH, W. D.: Goiter: from the Standpoint of Prevention 267

GORDON, A. H.: Goiter: Its Medical Aspect 267

MCGUFFIN, W. H.: Goiter: from a Radiologist's Viewpoint 267

FAHRNI, G. S.: Goiter: Its Surgical Treatment 267

MAYHEW, J. M.: The Basal Metabolism and the Blood Chemistry 268

EMERSON, C.: The Pathology of Goiter 268

BLISS, R. W.: The Medical Management of Goiter 268

ROWE, E. W.: The Roentgen-Ray Treatment of Goiter 268

HOTZ, G.: The Operative Treatment of Basedow's Disease 268

KORENCHEVSKY, V.: The Influence of the Removal of the Thyroid, Parathyroid, and Sexual Glands and of Thyroid Feeding upon the Regulation of the Body Temperature of Rabbits 269

SURGERY OF THE NERVOUS SYSTEM

Brain and Its Coverings; Cranial Nerves

GROVE, L. W.: Mixed Tumors of the Parotid Gland and Their Removal, with a Consideration of the Seventh Nerve 259

DEMEL: Living Dogs Whose Skulls Have Been Subjected to Roentgen Irradiation 270

SARGENT, P.: Types of Cerebral Tumors 270

SOUTTAR, H. S.: A New Form of Craniotome for Opening the Skull 270

BERTWISTLE, A. P.: Localization by the Roentgen-Rays 270

HEYMANN, E.: Clinical Experiences with the Development and Removability of Tumors at the Cerebellopontine Angle on the Basis of Twenty-Two Observations 271

EAGLETON, W. P.: Otitic Meningitis 271

BRUNNER, H.: Plastic Operations for Facial Paralysis 272

Peripheral Nerves

HERTZLER, A. E.: The Nature and Treatment of Sciatica 272

Sympathetic Nerves

ISAKOWITZ, H.: Injury of the Heart Muscle After Division of the Extracardial Nerves, and a Contribution on the Operative Treatment of Angina Pectoris 273

KUEMMELL: The Causes of Failure in Operations for Asthma and Their Prevention 273

DITTLER, R., and MUELLER, H. K.: Experiences with Regard to the Efficiency of the Vagus After Bilateral Extirpation of the Stellate Ganglion .. 273

SURGERY OF THE CHEST

Chest Wall and Breast

SWAN, R. H. J., and FRY, H. J. B.: Tuberculosis of the Male Breast 274

Trachea, Lungs, and Pleura

DESJARDINS, A. U.: The Reaction of the Pleura and Lungs to Roentgen Rays 274

Heart and Pericardium

ISAKOWITZ, H.: Injury of the Heart Muscle After Division of the Extracardial Nerves, and a Contribution on the Operative Treatment of Angina Pectoris 273

Œsophagus and Mediastinum

MAYDL, V.: A Case of Fatal Rupture of the Cardia in Dilatation for Cardiospasm with Stark's Sound 275

SURGERY OF THE ABDOMEN

Abdominal Wall and Peritoneum

SSOKOLOVSKIJ, M. P.: The Absorption of Bacteria from the Abdominal Cavity 276

KIRSCHNER: The Treatment of Suppurative Diffuse Peritonitis 276

PRIBRAM, B. O.: Mesenteric Lymphangeitis 281

Gastro-Intestinal Tract

KOENIG, F.: The Treatment of Gastric Ulcer 281

LINDBOE, E.: Experiences with the Resection Method of Pólya in Ulcer of the Stomach and Duodenum 282

DEAVER, J. B.: Intestinal Obstruction 282

KOERTE, W.: The Treatment of Acute Mechanical Occlusion of the Intestines 283

SCHMIEDEN: Precancerous Diseases of the Intestine, Especially in Polyposis 284

HARRINGTON, S.: Traumatic Retroperitoneal Rupture of the Duodenum; Traumatic Intraperitoneal and Extraperitoneal Rupture of the Duodenum; Strangulated Meckel's Diverticulum in the Right Femoral Canal; Solitary Non-Parasitic Cyst of the Liver 285

MOFFITT, H. C.: The Medical Aspects of Duodenal Ulcer 285

STARR, F. N. G.: The Surgical Treatment of Duodenal Ulcer 285

DICKSON, W. H.: The Radiological Aspect of Duodenal Ulcer 285

MAYO, C. H., and POWELL, L. D.: The Colon as a Urinary Receptacle 287

KELLING: The Question of Unsuccessful Appendectomies 288

CARLSON, G. W., and MARSHALL, V. F.: Intestinal Obstruction Following Appendectomy 288

LUNDY, J. S., and MCCUSKEY, C. F.: Sacral Anæsthesia in Operations on the Rectum 288

Liver, Gall Bladder, Pancreas, and Spleen

BOLLMAN, J. L., SHEARD, C., and MANN, F.C.: The Surgical Significance of Experimental Studies Concerning the Sites of Formation of Bile Pigment 289

WILDEGANS: Experimental and Clinical Investigations in Cholæmia 289

PANCOAST, H. K.: The Roentgenological Diagnosis of Liver Abscess with or without Subdiaphragmatic Abscess 290

MCINDOE, A. H.: Intrahepatic Lithiasis Associated with Multiple Internal Biliary Fistulæ 290

DUCHINOVA, S. I.: Temporary Clamping Off of the Hepatoduodenal Ligament for Bloodless Operations on the Liver 290

HIGGINS, G. M., and MANN, F. C.: Consideration of the Gall Bladder with Reference to the Process of Emptying 291

STEWART, W. H., and RYAN, E. J.: Further Advancements in the Technique and Interpretation of Cholecystography by the Oral Method 291

GRAHAM, E. A., COLE, W. H., COPHER, G. H., and KODAMA, S.: Some New Phases of the Physiology of the Biliary Tract 291

RICHARDSON, E. P.: Surgical Aspects of Certain Phases of Liver Function 291

MCGUIRE, E. R.: Problems in Gall-Bladder Surgery . 291

CAVE, H. W.: The Dangers Incident to Cholecystectomy 291

DEAVER, J. B., and BURDEN, V. G.: The Surgical Management of the Complications of Cholecystitis 291

BRUCE, H. A.: Association of Cholecystitis with Duodenal Ulcer 291

DOUGLAS, J.: Strictures and Operative Injuries of the Bile Ducts 291

CRILE, G. W.: The Operative Management of Common Duct Stones 291

CORBETT, R. S., and PEIRCE, C. B.: A Clinical Type of Cholelithiasis Resembling Renal Disease; Report of Two Cases 294

GYNECOLOGY

Uterus

MASSON, J. C.: Acute Inversion of the Uterus..... 295
WHITEHOUSE, B., and SHAW, W. F.: The Causes and Treatment of Uterine Hæmorrhage..... 295
PEMBERTON, F. A.: The Relation Between the Treatment of Cancer of the Cervix and the Cell Type..... 295

Adnexal and Periuterine Conditions

WHITEHOUSE, B.: The Influence of the Corpus Luteum upon Menstruation..... 296
MCCREADY, R. L., and RYAN, E. J.: Roentgenography of the Cavity of the Uterus and Fallopian Tubes, with Special Reference to Its Value in Cases of Sterility..... 297

External Genitalia

SHERMAN, E. M., and NORTON, S. L.: Further Research in the Problem of Vulvovaginitis in Children. II. Serological Studies..... 297

Miscellaneous

HUGGINS, R. R.: Ligation of Pelvic Veins in Thrombophlebitis..... 298
SAMPSON, J. A.: Endometriosis of the Sac of a Right Inguinal Hernia Associated with a Pelvic Peritoneal Endometriosis and an Endometrial Cyst of the Ovary..... 298
NOVAK, E.: The Significance of Uterine Mucosa in the Fallopian Tube, with a Discussion of the Origin of Aberrant Endometrium..... 298
DAVIS, C. H., and CRON, R. S.: A Contribution to the Study of Endometriosis..... 298

OBSTETRICS

Pregnancy and Its Complications

THOMSON, C. J.: The Causation of Still-Birth and Neonatal Death..... 301
EDEN, T. W.: The Indications for the Induction of Abortion..... 301
MCILROY, A. L.: Aspects To Be Considered for the Termination of Pregnancy Before Viability of the Child..... 301
BARRIS, J. D.: Slow and Rapid Methods of Induction of Abortion..... 301
PRICE, F. W.: The Cardiac Indications for the Termination of Pregnancy Before Viability of the Child..... 301
COLE, R. H.: The Plea of Insanity for the Termination of Pregnancy Before Viability of the Child. 301
EVERS, H. H.: The Ethical Aspect of Termination of Pregnancy Before Viability of the Child.... 301
SMITH, M. G.: On the Interruption of Pregnancy in the Rat by the Injection of Ovarian Follicular Extract..... 304

Labor and Its Complications

BAILEY, H.: Trial Labor in the Treatment of Four Hundred and Seventy-Seven Cases of Contracted Pelvis..... 304
BANISTER, J. B.: The Place of Induction of Premature Labor in the Treatment of Contracted Pelvis..... 305
SPALDING, A. B.: Limitations for the Cæsarean Operation..... 305
WILLIAMS, J. W.: Cæsarean Section at the Johns Hopkins Hospital..... 305

Puerperium and Its Complications

GOODMAN, S. J.: The Treatment of Puerperal Sepsis. 306

Newborn

BLANCO, L. V., and PAPERINI, H.: Meningeal Hæmorrhage in the Newborn..... 306

GENITO-URINARY SURGERY

Adrenal, Kidney, and Ureter

CORBETT, R. S., and PEIRCE, C. B.: A Clinical Type of Cholelithiasis Resembling Renal Disease; Report of Two Cases..... 294
WILLIAMSON, C. S.: Further Studies on the Transplantation of the Kidney..... 307
FEY, B.: The Functional Results of Retention in the Renal Pelvis..... 307
BEER, E.: Uric Acid and Uratic Stones in the Kidney—Uric Acid Showers and Their Diagnosis..... 310
ROSENO, A.: Postoperative Hæmorrhage Following Nephrotomy and Its Prevention..... 311
LIPSHUTZ, B., and HOFFMAN, C.: Renal Arterial Variations and Extraperitoneal Abdominal Nephrectomy..... 311
JOSEPH, E.: The Removal of Deeply Situated Ureteral Calculi..... 311

Bladder, Urethra, and Penis

COLBY, F. H.: Bladder Symptoms from Congenital Deformities with Associated Nerve Lesions: Report of Three Cases..... 312

Genital Organs

ZENTMAYER, W.: The Prostate as a Remote Focus of Infection in Ocular Inflammations..... 260
FRONSTEIN, R., and MESCHEBOWSKI, G.: Recurrences Following Prostatectomy by the Method of Freyer..... 312

Miscellaneous

HINMAN, F., and REDEWILL, F. H.: Pyelovenous Back Flow..... 313
MIXTER, C. G.: Urinary Obstructions Occurring in Childhood..... 313

HYMAN, A.: The Clinical Aspects of Surgical Diseases of the Urinary Tract in Children..... 314
MACKENZIE, D. W.: Malignant Growths of the Lower Urinary Tract... 315
MANN, L. T.: The Results of X-Ray Therapy of Malignant Growths of the Urinary Tract..... 315

SURGERY OF THE BONES, JOINTS, MUSCLES, TENDONS

Conditions of the Bones, Joints, Muscles, Tendons, Etc.

NEW, G. B., and DECKER, W. J.: Pharyngeal Sinus with Cervical Pott's Disease; Report of Six Cases..... 265
KRASNOBAJEV, T. P.: The Treatment of Acute Hæmatogenous Infectious Osteomyelitis..... 316
MEYERDING, H. W.: Chronic Infectious Arthritis with Multiple Ankylosis of Joints and Complete Disability..... 316
SEELIGER: The Pathological Physiology of the Joints; The Formation of Joint Mice..... 316
LOEHR, W.: Ischæmic Contracture..... 317
HICKEY, P. M.: X-Ray Clinic on Lesions of the Vertebræ..... 317
KONJETZNY: A Contribution to Our Knowledge of Perthes' and Koehler's Disease..... 319
JUENGLING, O.: The Results of the Roentgen Treatment of Bone and Joint Tuberculosis in the Region of the Foot.. 319

Surgery of the Bones, Joints, Muscles, Tendons, Etc.

PAYR: Plastic Operations on the Large Ball and Socket Joints..... 320
WHITMAN, A.: Remarks Prefatory to a Cinematographic Presentation of Late Results of the Reconstruction Operation..... 321
ABBOTT, L. C.: The Correction of Deformity in Quiescent Disease of the Hip..... 321

Fractures and Dislocations

HENDERSON, M. S.: The Cause and Treatment of Ununited Fractures..... 322
EDWARDS, H. C.: The Mechanism and Treatment of Backfire Fracture..... 322
CLARK, W. A.: A Light Portable Extension Frame.. 323

SURGERY OF BLOOD AND LYMPH SYSTEMS

Blood Vessels

LIPSHUTZ, B., and HOFFMAN, C.: Renal Arterial Variations and Extraperitoneal Abdominal Nephrectomy 311
FRY, H. J. B., and SHATTOCK, C. E.: Sarcomatous Parmeation of the Inferior Vena Cava and the Right Side of the Heart..... 324

Blood; Transfusion

MAYHEW, J. M.: The Basal Metabolism and the Blood Chemistry..... 268
MAYO, W. J.: Dyscrasias of the Blood 324
SPEESE, J.: The Surgical Aspect of Blood Dyscrasias. 325
ROLLESTON, SIR H.: Indications for Blood Transfusion..... 325
CRUCHET, R.: Transfusion of Blood from Animal to Man..... 325
KEYNES, G.: Blood Transfusion in Surgery..... 325
GRULEE, C. G.: Intraperitoneal Transfusion in von Jaksch's Anæmia..... 326

SURGICAL TECHNIQUE

Operative Surgery and Technique; Postoperative Treatment

REHN, E.: Surgery and Organ Function..... 327

Anæsthesia

LUNDY, J. S., and MCCUSKEY, C. F.: Sacral Anæsthesia in Operations on the Rectum..... 288

PHYSICOCHEMICAL METHODS IN SURGERY

Roentgenology

MCGUFFIN, W. H.: Goiter: from a Radiologist's Viewpoint..... 267
ROWE, E. W.: The Roentgen-Ray Treatment of Goiter 268
DEMEL: Living Dogs Whose Skulls Have Been Subjected to Roentgen Irradiation..... 270
BERTWISTLE, A. P.: Localization by the Roentgen-Rays 270
DESJARDINS, A. U.: The Reaction of the Pleura and Lungs to Roentgen Rays..... 274
DICKSON, W. H.: The Radiological Aspect of Duodenal Ulcer..... 285
PANCOAST, H. K.: The Roentgenological Diagnosis of Liver Abscess with or without Subdiaphragmatic Abscess..... 290
STEWART, W. H., and RYAN, E. J.: Further Advancements in the Technique and Interpretation of Cholecystography by the Oral Method.. 291
MCCREADY, R. L., and RYAN, E. J.: Roentgenography of the Cavity of the Uterus and Fallopian Tubes, with Special Reference to Its Value in Cases of Sterility..... 297
MANN, L. T.: The Results of X-Ray Therapy of Malignant Growths of the Urinary Tract..... 315
HICKEY, P. M.: X-Ray Clinic on Lesions of the Vertebræ. 317
JUENGLING, O.: The Results of the Roentgen Treatment of Bone and Joint Tuberculosis in the Region of the Foot..... 319
PUTNAM, T. J.: Some Brominized Oils for Radiographic Use: Preliminary Report..... 329
DESJARDINS, A. U.: Stimulation and Immunity in Radiotherapy..... 329

Radium

MCGUFFIN, W. H.: Goiter: from a Radiologist's Viewpoint..... 267

Miscellaneous

MERTZ, A.: An Increase of Ferment in the Serum Following Light and Roentgen-Ray Irradiation, an Index of the Processes of Cell Destruction 329
HESS, A. F.: The Use and Misuse of Ultraviolet Therapy 330
PAÏZIS, D.: The Treatment of Moist Gangrene in Diabetics by Diathermy 330

MISCELLANEOUS

Clinical Entities—General Physiological Conditions

MENNE, F. B., JOYCE, T. M., and VON HUNGEN, A. P.: Thyroid Disturbances: A Clinicopathological Study of 300 Instances 266
KEITH, W. D.: Goiter from the Standpoint of Prevention 267

General Bacterial, Protozoan, and Parasitic Infections

MILLS, L.: Ocular Disease Occurring in the Course of Non-Dysenteric Amœbiasis 260
KAISERLING: Sepsis from the Pathologico-Anatomical Standpoint 331
NICKEL, A. C.: The Localization in Animals of Bacteria Isolated from Foci of Infection 331
JACKSON, R. H.: Surgical Treatment of Certain Massive Blastomycotic Skin Lesions 331

Ductless Glands

ROWNTREE, L. G., and SNELL, A. M.: The Diagnosis and Treatment of Certain Glandular Deficiencies 332

BIBLIOGRAPHY

Surgery of the Head and Neck

Head 333
Eye 333
Ear 334
Nose and Sinuses 334
Mouth 335
Pharynx 335
Neck 335

Surgery of the Nervous System

Brain and Its Coverings; Cranial Nerves 336
Spinal Cord and Its Coverings 337
Peripheral Nerves 337
Sympathetic Nerves 337
Miscellaneous 338

Surgery of the Chest

Chest Wall and Breast 338
Trachea, Lungs, and Pleura 338
Heart and Pericardium 339
Œsophagus and Mediastinum 339
Miscellaneous 339

Surgery of the Abdomen

Abdominal Wall and Peritoneum 339
Gastro-Intestinal Tract 340
Liver, Gall Bladder, Pancreas, and Spleen 343
Miscellaneous 344

Gynecology

Uterus 345
Adnexal and Periuterine Conditions 345
External Genitalia 346
Miscellaneous 346

Obstetrics

Pregnancy and Its Complications 347
Labor and Its Complications 349
Puerperium and Its Complications 350
Newborn 350
Miscellaneous 350

Genito-Urinary Surgery

Adrenal, Kidney, and Ureter 351
Bladder, Urethra, and Penis 352
Genital Organs 352
Miscellaneous 352

Surgery of the Bones, Joints, Muscles, Tendons

Conditions of the Bones, Joints, Muscles, Tendons, Etc. 353
Surgery of the Bones, Joints, Muscles, Tendons, Etc. 354
Fractures and Dislocations 355
Orthopedics in General 352

Surgery of the Blood and Lymph Systems

Blood Vessels 351
Blood; Transfusion 357
Lymph Vessels and Glands 357

Surgical Technique

Operative Surgery and Technique; Postoperative Treatment 357
Antiseptic Surgery; Treatment of Wounds and Infections 358
Anæthesia 358

Physicochemical Methods in Surgery

Roentgenology 359
Radium 359
Miscellaneous 359

Miscellaneous

Clinical Entities—General Physiological Conditions 359
General Bacterial, Mycotic, and Protozoan Infections 360
Ductless Glands 360
Surgical Pathology and Diagnosis 360
Experimental Surgery 360
Hospitals; Medical Education and History 360

AUTHORS

OF THE ORIGINAL ARTICLES ABSTRACTED IN THIS NUMBER

Abbott, L. C., 321
Bailey, H., 304
Banister, J. B., 305
Barris, J. D., 301
Beer, E., 310
Bertwistle, A. P., 270
Biehl, C., 262
Blanco, L. V., 306
Bliss, R. W., 268
Bollman, J. L., 289
Brown, E. V. L., 260
Brown, G. V. I., 264
Bruce, H. A., 291
Brunner, H., 272
Burden, V. G., 291
Carlson, G. W., 288
Cave, H. W., 291
Clark, W. A., 323
Colby, F. H., 312
Cole, R. H., 301
Cole, W. H., 291
Copher, G. H., 291
Corbett, R. S., 294
Crile, G. W., 291
Cron, R.S., 298
Cruchet, R., 325
Davis, C. H., 298
Deaver, J. B., 282, 291
Decker, W. J., 265
Demel, 270
Desjardins, A. U., 274, 329
Dickson, W. H., 285
Dittler, R., 273
Dixon, O. J., 259
Douglas, J., 291
Duchinova, S. I., 290
Eagleton, W. P., 271
Eden, T. W., 301
Edwards, H. C., 322
Emerson, C., 268
Evers, H. H., 301
Fahrni, G. S., 267
Fey, B., 307
Figi, F. A., 265
Fronstein, R., 312
Fry, H. J. B., 274, 324
Goldstein, M. A., 261
Goodman, S. J., 306
Gordon, A. H., 267
Graham, E. A., 291
Grove, L. W., 259
Grulee, C. G., 326
Harrington, S., 285
Henderson, M. S., 322
Hertzler, A. E., 272
Hess, A. F., 330
Heymann, E., 271
Hickey, P. M., 317
Higgins, G. M., 291
Hinman, F., 313
Hoffman, C., 311
Hotz, G., 268
Hudson, R. V., 265
Huggins, R. R., 298
Hyman, A., 314
Isakowitz, H., 273
Irons, E. E., 260
Jackson, R. H., 331
Joseph, E., 311
Joyce, T. M., 266
Juengling, O., 319
Kaiserling, 331
Keith, W. D., 267
Kelling, 288
Keynes, G., 325
Kirschner, 276
Kodama, S., 291
Koenig, F., 281
Koerte, W., 283
Konjetzny, 319
Korenchevsky, V., 269
Krasnobajev, T. P., 316
Kuemmell, 273
Leidler, R., 262
Lillie, H. I., 263
Lindboe, E., 282
Lipshutz, B., 311
Loehr, W., 317
Lundy, J. S., 288
MacKenzie, D. W., 315
Mann, F. C., 289, 291
Mann, L. T., 315
Marshall, V. F., 288
Masson, J. C., 295
Maydl, V., 275
Mayhew, J. M., 268
Mayo, C. H., 287
Mayo, W. J., 324
McCready, R. L., 297
McCuskey, C. F., 288
McGuffin, W. H.,,267
McGuire, E. R., 291
McIlroy, A. L., 301
McIndoe, A. H., 290
McMahon, B. J., 264
Mertz, A., 329
Meschebowski, G., 312
Meyerding, H. W., 316
Mills, L., 260
Mixter, C. G., 313
Moffitt, H. C., 285
Mueller, H. K., 273
New, G. B., 265
Nickel, A. C., 331
Norton, S. L., 297
Novak, E., 298
Païzis, D., 330
Pancoast, H. K., 290
Paperini, H., 306
Payr, 320
Peirce, C. B., 294
Pemberton, F. A., 295
Powell, L. D., 287
Pribram, B. O., 281
Price, F. W., 301
Putnam, T. J., 329
Redewill, F. H., 313
Rehn, E., 327
Richardson, E. P., 291
Rolleston, Sir H., 325
Roseno, A., 311
Rowe, E. W., 268
Rowntree, L. G., 332
Ryan, E. J., 291, 297
Sampson, J. A., 298
Sargent, P., 270
Schild, E. H., 260
Schmieden, 284
Seeliger, 316
Shattock, C. E., 324
Shaw, W. F., 295
Sheard, C., 289
Sherman, E. M., 297
Smith, M. G., 304
Snell, A. M., 331
Souttar, H. S., 270
Spalding, A. B., 305
Speese, J., 325
Ssokolovskij, M. P., 276
Stammers, F. A. R., 259
Stark, W. B., 263
Starr, F. N. G., 285
Stewart, W. H., 291
Swan, R. H. J., 274
Tait, J., 261
Thompson, J. E., 264
Thomson, C. J., 301
Wildegans, 289
Willcox, Sir W., 263
Whitehouse, B., 296
Whitman, A., 321
Williams, J. W., 305
Williamson, C. S., 307
Zentmayer, W., 260

EDITOR'S COMMENT

THREE papers of particular interest from European clinics, by Kirschner on peritonitis (p. 276), by Koerte on intestinal obstruction (p. 283), and by Fey on retention in the kidney pelvis (p. 307), are reviewed at length in this month's issue of the ABSTRACT. Kirschner's analysis of the results of thirty years' treatment of peritonitis at the Koenigsberg Clinic and of the reports received from twenty-three other clinics and hospitals, his conclusions as to the most effective method of treatment, and the extended discussion of his paper by representatives of most of the surgical clinics of central Europe deserve careful reading.

Many interesting observations with reference to treatment are mentioned in the discussion—the effect of irrigation with various solutions upon different types of infection and upon the intestinal movements and circulation, the efficacy of an intestinal fistula and the proper time for its formation, and the futility of attempting to drain the peritoneal cavity.

A few sentences from Kirschner's paper deserve to be emphasized, "In laparotomies the chief essential is gentleness in the handling of the tissues. . . . The main object of interference is the certain removal of the source of infection in the simplest manner. . . . With regard to the manner of removing exudate found in the abdominal cavity, whether by irrigation or by dry sponging . . . . statistics do not show any marked difference in the results."

He believes that drugs should not be introduced into the peritoneal cavity and that drainage of the free abdominal cavity should not be attempted. He feels that the theoretical advantages of Fowler's position after operations are outweighed by the discomfort it causes the patient. Morphine, the maintenance of fluid intake, gastric lavage, and glucose with insulin, are stressed as important factors in the successful postoperative care.

In the treatment of acute mechanical obstruction, Koerte emphasizes the very great importance of early surgical intervention and of "methodical emptying of the over-filled intestine in an otherwise conservative and faultlessly aseptic procedure."

By filling the kidney pelvis with sodium iodide solution and observing the changes in contour of the pelvis and ureter under the fluoroscopic screen, Fey and his coworkers have attempted to determine the appearance and form of the kidney pelvis at different times and under varying conditions, and thus arrive at a classification of the causes and types of retention in the kidney pelvis, and of the indications for and the results of treatment.

In discussing the cause and treatment of ununited fractures, Henderson (p. 322) calls attention again to the work of Robison with reference to the presence of an enzyme in the osteoblasts and cartilage cells which acts in the phosphoric esters of the blood and whose action is inhibited by the amino acids formed in the breaking down of muscle tissue and blood clot. He suggests that Robison's work, if accepted, offers a logical basis for the treating fractures complicated by extensive injury of the soft parts by open operation, with careful removal of damaged tissue and blood clots and accurate reduction.

Sargent's encouraging statistics with reference to the prognosis in cerebral tumors (p. 270), Beer's discussion of the symptoms and recognition of uric acid stones in the kidney and of uric acid showers (p. 310), Bollman, Sheard and Mann's experimental studies on the origin of bile pigment (p. 289), and Putnam's investigation of various oils and halogen derivatives with reference to their suitability for radiographic use (p. 328) are only a few of many other important abstracts in this month's issue of the INTERNATIONAL ABSTRACT OF SURGERY.

INTERNATIONAL ABSTRACT OF SURGERY

APRIL, 1927

ABSTRACTS OF CURRENT LITERATURE

SURGERY OF THE HEAD AND NECK

HEAD

Dixon, O. J.: The Pathological Examination in Cavernous Sinus Thrombosis as a Guide to the Diagnosis, Prognosis, and Treatment. *J. Am. M. Ass.*, 1926, lxxxvii, 1088.

The author reports eight cases of cavernous sinus thrombosis following: (1) trauma to a pustule on the upper lip, (2) the incision of a peritonsillar abscess, (3) a septic sore throat, (4) a postoperative mastoid infection by way of the sigmoid, petrosal, and cavernous sinuses, (5) curettage of a tooth socket, (6) acute osteomyelitis of the mandible, (7) the extraction of a tooth, and (8) supra-orbital cellulitis. From these cases the following conclusions are drawn:

1. The primary foci are usually ordinary and trivial infections.
2. Some form of trauma is usually associated with these primary infections.
3. The thrombosis takes place by direct extension of the thrombophlebitis.
4. The offending organism is usually the staphylococcus.
5. The temperature is sustained and gradually increases. Chills are usually absent.
6. Meningitis follows so closely after the development of the eye symptoms that septic thrombosis of the cavernous sinus is a non-surgical complication.
7. Recoveries which have been reported probably occurred in cases with an erroneous diagnosis.
8. Meningitis and septicæmia do not explain the rapid termination.

MANFORD R. WALTZ, M.D.

Stammers, F. A. R.: Pre-Auricular Fistulæ. *Brit. J. Surg.*, 1926, xiv, 359.

The author reports six cases of pre-auricular fistula which showed a familial and hereditary tendency.

Because the disfigurement is negligible and because of the absence of symptoms unless complications develop, very little notice has been taken of this congenital deformity. Sometimes, however, an ulcer develops in front of the ear as the result of blocking of the small sinus, and this leads to an investigation of the ear.

There are three theories regarding the etiology of the deformity. According to one, such a fistula is the remains of the first branchial cleft. According to another, it is due to failure of union between two of the six tubercles which appear around the posterior end of the first cleft and go to form the pinna, or to some anomalous process occurring in connection with these tubercles. According to the third theory, the fistula is due to the failure of union of the soft parts corresponding to, and overlying, the posterior ends of the mandibular and maxillary processes of the first arch.

The majority of textbooks still favor the first of these three theories, but Stammers presents evidence which has led him to the conclusion that the second theory is the most plausible.

JACOB S. GROVE, M.D.

Grove, L. W.: Mixed Tumors of the Parotid Gland and Their Removal, with a Consideration of the Seventh Nerve. *South. M. J.*, 1926, xix, 752.

It was formerly believed that mixed tumors of the parotid are epithelial in type, springing from the ducts or acini, and that trauma, such as that which may be caused by mastication is an exciting factor in their development.

New has shown that no direct connection can be demonstrated between the gland and the growth, and that, from the very beginning, these growths are thoroughly encapsulated. They may develop from encapsulated cell rests. If trauma were a factor, they would be more common in the submaxillary gland.

Volkman and DeCosta believe that they are derived from the endothelium of the blood vessels and lymphatics.

They are rare. They begin as small painless growths, usually manifested in the second decade

of life and growing slowly. A large percentage enlarge with breaking down of the capsule and general invasion of the gland. In 25 per cent of Wood's forty-nine cases, the tumor became malignant and in 45 per cent a recurrence followed the removal of the malignant growth. Complete removal while the tumor is still encapsulated will result in a cure in practically every case (Sistrunk).

Under local anæsthesia, Grove makes an incision along the horizontal branches of the facial nerve, isolating the nerve branches and continually keeping them under view during the dissection of the tumor. He reports three cases.

In the first case a tumor the size of a hen's egg had been present for twenty years. It was firmly fixed in front of the right ear and in the past six months had increased in size. In the second case there was a tumor the size of an almond, of six months' duration, and in the third case, a small tumor of one year's duration which had grown rapidly during the past six months.

These growths were all enucleated under local anæsthesia by incisions along the branches of the seventh nerve, the nerve being carefully isolated and kept in view during the dissection. No facial paralysis resulted from the procedure.

HARRY C. SALTZSTEIN, M.D.

EYE

Schild, E. H.: An Unlearnable Prism Test for Suspected Malingering. *Am. J. Ophth.*, 1926, 3 s. ix, 741.

The prism used in the test described by Schild is a small piece of rectangular glass measuring about 1 by 2 in., one-half of which is a rather thick plano and near the center tapers off to a prism of 5 degrees for the other half. It is very important that the base line of the prism which runs across the middle of the glass should be as sharp as possible to make an abrupt change to the prism side.

A suitable test object is provided. Ordinarily any small bright object against a plain background will do except an electric light bulb or other bright light. A small white visiting card is best.

With this glass one may produce the effect of either a plano, a double prism, or a single 5-degree prism, depending upon the way it is held. If it is held so that the prism end is up and the dividing line is just above the pupillary border, the view will be through the plano part. This is Position 1. By lowering it so that the dividing line runs midway across the pupil, the effect of a double prism is obtained. This is Position 2. Lowering it again so that the dividing line is below the edge of the pupil gives the effect of a single 5-degree prism with the single image displaced upward. This is Position 3. The shifting from Position 2 to Position 3 is the critical stage of the test and must be done at the moment when the subject has both eyes open and his attention distracted so that he will not notice the change.

The examination deals entirely with the good eye. If the patient shows signs of memorizing his replies, the position of the glass must be secretly reversed.

L. L. MCCOY, M.D.

Irons, E. E., and Brown, E. V. L.: Recurrence of Iritis as Influenced by the Removal of Infections: Summary of Fifty Cases. *J. Am. M. Ass.*, 1926, lxxxvii, 1167.

Zentmayer, W.: The Prostate as a Remote Focus of Infection in Ocular Inflammations. *J. Am. M. Ass.*, 1926, lxxxvii, 1172.

Mills, L.: Ocular Disease Occurring in the Course of Non-Dysenteric Amœbiasis. *J. Am. M. Ass.*, 1926, lxxxvii, 1176.

IRONS and BROWN report the late results in fifty cases of iritis from three to twelve years after treatment. In forty-three there had been no recurrence.

In seventeen of the cases the iritis was attributed to tonsillar infection, and in thirteen of these the tonsils were removed. In ten cases, infected teeth were believed responsible and were extracted. In two cases, anti-syphilis treatment was given, and in two others, tuberculin was used. In the remaining cases the underlying condition was a genito-urinary infection or a combination of various conditions with tonsil and dental infection or was not determined.

Of the seven patients in whom the iritis recurred, one refused to allow the removal of badly infected tonsils. In the case of another, treatment for lues was given and an infected tooth extracted, but infected tonsils were not removed. Two of the patients with recurrences had gonorrhœal prostatitis and arthritis. A fifth patient had a generalized infection associated with sinusitis and the sixth an apparently healed pulmonary tuberculosis and a severe episcleritis. Both of these developed keratitis. The seventh reacted to tuberculin, but refused to remain under observation for a sufficient length of time for adequate study.

ZENTMAYER reports in detail four cases of ocular inflammation in which the prostate seemed to be the source of the infection.

In the first case there had been repeated attacks of gonorrhœal arthritis and iritis. The sight of the right eye had been lost as the result of secondary glaucoma. In the left eye, in spite of an iridectomy during an attack of acute glaucoma, vision had decreased to 1/70. Prostatic massage and the use of stock and autogenous vaccines seemed to make the eye condition worse.

The second case was that of a man 50 years old who had prostatitis. On two occasions in this case prostatic massage was followed by an attack of acute iritis.

The third case was that of a man 26 years old who gave a history of gonorrhœal urethritis and arthritis nine years before the iritis. Four years later he had a very severe recurrence of iridocyclitis, and a diagnosis of prostatitis was made.

The fourth case was one of central exudative retinochoroiditis occurring in a man who showed no other lesion except prostatitis.

Zentmayer cites also three cases of vesicular keratitis in men with a history of gonorrhœa. He draws the following conclusions:

1. The prostate may be the source of infection in certain ocular inflammations.

2. The prostatic infection is usually non-gonococcal.

3. The metastasis, as in other focal infections, may occur in any of the ocular tissues susceptible to inflammatory reaction, but the uvea and cornea are probably involved most often.

4. The fact that an inflammation persists after the removal of a suspected focus does not prove that this focus was not the primary source of the infection; the inflammation may have so reduced the resistance of the tissues as to render them unable to withstand the action of much less virulent organisms or toxins from some other part of the body.

5. Only the subsidence of a metastatic inflammation after the removal of a possible source of infection proves that this source was the exciter of the condition.

Mills reports eight cases of ocular inflammation associated with intestinal amœbiasis in which the eye condition was relieved by the treatment of the amœbiasis. He believes that many more cases would be found if laboratory workers were familiar with intestinal protozoa. The conditions in the cases reported were iritis associated with migraine-like headache and gastro-intestinal symptoms; iridocyclitis persisting for eighteen months and associated with arthritis and a history of gonorrhœal infection; bilateral iritis persisting for ten months; bilateral iritis persisting for five years; severe conjunctivitis and episcleritis of eleven weeks' duration; recurring attacks of sclerosing keratitis for six years; iritis persisting for six years and causing blindness from secondary glaucoma; and choroidal atrophy of one eye and recurrent iritis in the other eye.

Also reported are three cases of retinal hæmorrhage, one case of incipient cataract, two cases of glaucoma simplex, and three cases of retinal detachment which were favorably influenced by the treatment of associated amœbiasis.

Mills agrees with Malling that many cases of so-called primary glaucoma are in fact secondary, as is often shown by the findings made with the slit lamp. He has noticed that synechiæ are frequently not marginal but arise from the surface of the iris or the ciliary body.

A large percentage of chronic ocular conditions which resist local treatment are associated with intestinal infection by protozoa or flagellates causing colitis. In most of such ocular conditions, treatment for the amœbiasis has resulted in relief, arrest, or cure of the eye condition. Emetin may be of value also in iritis due to other causes. Any chronic or recurrent or intractable ocular disease in which the elimination of focal infection and correction of the diet is not of benefit, should be treated as a parasitic intestinal infection. Samuel A. Durr, M.D.

EAR

Goldstein, M. A.: The Relation of Tactile Impression and Hearing Perception. *Laryngoscope*, 1926, xxxvi, 729.

Goldstein traces the development of the sensory organs in animals from the lower to the higher types. The embryological derivation of the auditory organ is considered. The purpose of the discussion is to outline embryologically and phylogenetically the relationship and gradual development of the general touch sense to its most complicated mechanism of special sensory organs and especially to the auditory organ in the mammalia and in the human species.

The lower the anatomical, physiological, and economic zoological rank of the animal the simpler and more rudimentary are its sensory organs.

The statement has not yet been challenged that light, heat, sound, and electricity are modifications of the same wave of motion, varying in intensity, quality, and direction. In functional hearing tests of both the normal and the deaf subject it has been very difficult to determine where an auditory impression ceases and where a tactile impression begins, or to what degree one sensory impression may be translated into terms of the other.

Goldstein cites the case of a congenitally deaf girl who became so highly sensitized that she was able, blindfolded, to receive and repeat entire sentences heard through an ordinary megaphone, the distal end of which was spanned by a tense diaphragm of Whatman paper with which the tips of her fingers were in contact.

In conclusion the author states that there is much promise in the use of apparatus embodying radio and telephone principles for the amplification of sound. A. R. Hollender, M.D.

Tait, J.: Ablation Experiments on the Labyrinth of Frogs. *Laryngoscope*, 1926, xxxvi, 713.

In his experimental work on the frog the author observed that when one or both saccular otoliths are removed or when the nerves to the saccular maculæ are cut, the result so far as equilibrium is concerned is very clear and definite. The animal sits, crawls, jumps, swims absolutely normally. These negative results in normal behavior confirm previous work on fish by Parker and Maxwell and on frogs by Laudenbach which showed that the saccular macula, at least of these lower forms of animal life, is not in any way concerned with equilibrium.

The utricular macula is wholly an organ of static equilibrium. When the frog assumes stationary postures on an inclined plane, the head tends to be kept horizontal while the center of gravity of the body is plumbed within the base of support formed by the four limbs. These stationary postures, which vary with the degree of inclination of the plane on which the frog rests, constitute reactions of static equilibrium. In taking up the appropriate pose the

animal depends on nervous messages derived from four possible sources: (1) pressure impulses due to contact with the ground, (2) impulses from muscles and joints, (3) impulses from the eyes, and (4) impulses from the utricular maculæ.

So far as the body and limbs of the frog are concerned, the saccular macula has nothing to do with equilibrium. The utricular macula is the organ of static equilibrium and is concerned in the adoption and maintenance of appropriate postures in response to gravity.

The semicircular canals are for kinetic equilibrium. The static reactions are manifested only when the animal stands still.

Upon semicircular function depend appropriate responses to counteract sudden tiltings and jerkings. The canals are called into action by something sudden; their effect is momentary and evanescent. The plumb-line mechanism responds to a steady field of force; its action is sluggish, but its effect is sustained. This fact is somewhat reminiscent of the difference between an induced and a constant electric current.

The differential diagnosis of lesions of the canals depends upon a knowledge of their individual function.

Bárány's tests are reviewed. Bárány's method proves that stimulation of the canals is due to movement of the endolymph within them, but is complicated and not wholly satisfactory.

The horizontal canals are brought into action in rapid turning movements about a vertical axis. Experiments have proved that when the normal frog is quickly rotated toward the right it is the horizontal canal on that side that is stimulated. If the lesion is on the left side, the frog will fail to respond when turned toward the left.

The vertical canals are brought into operation in rapid tilting movements about horizontal axes. From experiments with frogs the conclusion is drawn that each vertical canal is especially associated with the limb of that quarter of the body toward which it points. It was noted also that whereas a vertical canal is specially associated with one particular limb, stimulation of a horizontal canal leads to movements of at least two and usually of all four limbs.

Deaf-mutes on the Bárány chair have no trouble in detecting even a low rate of angular acceleration and in indicating its direction. Occasionally, however, one finds a patient who shows obvious disability; his threshold is high and his answers are incorrect. Two such patients were examined. Both had acquired bilateral labyrinth trouble as a result of meningitis. These patients, when tested on the tilt-table, showed a behavior analogous to that of a completely decanalized frog. Other deaf-mutes who were examined reacted on the tilt-table in the same way as normal persons. When a patient was found defective by the very simple Bárány test he showed disability also on the tilt-table.

A. R. HOLLENDER, M.D.

Biehl, C.: Perforation of the Fenestra Rotunda for Therapeutic Purposes. *J. Laryngol. & Otol.*, 1926, xli, 637.

Leidler, R.: The Indications for Opening the Labyrinth. *J. Laryngol. & Otol.*, 1926, xli, 641.

In discussing the function of the fenestra rotunda BIEHL calls attention to the fact that the tympanic membrane and the obturator of the fenestra ovalis, the plate of the stapes, have a muscular movement, whereas the movement of the membrane of the fenestra depends on changes within the labyrinth, changes of pressure. Hence the fenestra rotunda is influenced by the quantity of fluid in the labyrinth and the reactions which these fluids undergo. It is certain that an increase of pressure in the labyrinth can be diminished by an operation on the fenestra ovalis.

When the fenestra rotunda is perforated, the increase of fluid, of pressure altogether, will have its effect on the vestibular side and will be manifested there only if the membrane of the fenestra cannot produce the effect.

The function of the membrane of the fenestra rotunda is twofold—adjustment of the mechanical effect, which requires a certain amount of flexibility, and adjustment of the acoustic effect which requires a certain rigidity with regard to these extremes.

In conjunction with the vestibular apparatus, the fenestra rotunda has a mechanical action, but in conjunction with the cochlear apparatus its action is an acoustic tension which will be at its strongest if the membrane is taut. In addition to these functions the fenestra rotunda has, by reason of its fibrous membranous structure, the capacity to diffuse and in this way to influence the process within the labyrinth.

Disturbances in the internal ear caused by overpressure can be removed or diminished only by perforating the fenestra rotunda.

LEIDLER agrees that the question as to when we ought to operate on the labyrinth is still far from satisfactorily answered, but states that in his opinion operation is indicated when the patient is suffering from chronic or acute otitis media with symptoms of diffuse acute inflammation of the labyrinth, that is to say, deafness, nystagmus of the third degree on the healthy side, and lack of response to the turning, caloric, and fistula tests, and at the same time there are symptoms of intracranial complications (a temperature over 38 degrees C., headache, and possibly positive signs in the cerebrospinal fluid). It is indicated also in cases of acute or chronic otitis media in which the labyrinth is completely deranged functionally and an antrotomy or radical operation is called for.

When facial paralysis occurs in the course of the disease, the cavities of the middle ear must be totally opened (radical operation). When disease of the labyrinth capsule is diagnosed (cholesteatoma, granulations in the windows, fistula), opening of the labyrinth is to be recommended even if the latter is still functioning.

When, in spite of removal by operation of middle ear suppuration and correspondingly long observation, a circumscribed disease of the labyrinth shows a tendency to increase (gradual loss of function, possibly accompanied by giddiness and headache, poor healing of the cavity) the labyrinth should be opened before the functions are wholly lost.

In a case of acute elimination of the eighth nerve (when it is probably impossible to decide whether the case corresponds to a serous or to a suppurating inflammation in the labyrinth), and it is necessary to operate for other reasons, the complete radical operation should be done on the middle ear even in acute cases in order that the lateral labyrinthine wall may be inspected.

In cases of abscess of the brain (especially with abscess of the cerebellum, and possibly with deep extradural abscess of the posterior fossa) the sacrifice of even an intact labyrinth may be necessary for exposure of the abscess. A. R. HOLLENDER, M.D.

Lillie, H. I., and Stark, W. B.: The Insidious, Symptomless, Destructive Effect of Cholesteatoma. *Surg. Clin. N. Am.*, 1926, vi, 1359.

Two interesting cases illustrating the insidious, symptomless, destructive effect of cholesteatomatous masses in the temporal bone have been observed in the Mayo Clinic.

The first case was that of a woman 26 years of age who complained of severe pain in the left ear, vertigo, and vomiting of three days' duration. Fifteen years previously she had had an attack of acute purulent otitis media on the left side. The tympanic membrane was thickened and bulging. The hearing was diminished, but the responses of the semicircular canals to stimulation were prompt. The roentgenogram showed a sclerotic mastoid.

Incision of the tympanic membrane was followed by immediate and complete relief of the pain and, after the lapse of a few hours, by cessation of the vertigo. There was a foul-smelling discharge. Diffuse labyrinthitis developed, but the acute symptoms subsided in a few days. At operation, it was found that the tympanic membrane and middle ear were filled with a cheesy cholesteatomatous mass. When this was removed, fistulæ were discovered leading to the superior and horizontal semicircular canals. Labyrinthectomy was performed. Uneventful recovery resulted.

The interesting features of this case were: the history of an old otitis media, all but forgotten by the patient; the intact tympanic membrane; the foul-smelling discharge following myringotomy; and the labyrinthine symptoms and findings.

The second case was that of a man aged 55 years. The only complaint was itching in the right ear. There was a small crust attached to the posteroinferior wall of the right external auditory canal just external to the isthmus. When this was removed a fistula was found leading into the mastoid. On being questioned, the patient recalled having had in childhood, an acute otitis media on the same side. The tympanic membrane, the hearing, and the responses of the semicircular canals to stimulation were normal. A roentgenogram revealed an extensive destructive lesion of the pars squamosa.

At operation, an unusually destructive process was found. The cholesteatomatous mass involved the squamous portion of the temporal bone to its limits and the petrous portion to the apex. It had dissected the capsule of the labyrinth, distinctly outlining it. The dura near the apex of the petrous portion was very thin, and the removal of the mass injured the dura, allowing the escape of cerebrospinal fluid, which continued to ooze for seventy-two hours. Convalescence was protracted, but no untoward symptoms developed.

These cases show how insidious and extensive may be the effects of cholesteatoma in the temporal bone in the absence of symptoms. In both cases there was a history of disease of the middle ear and the tympanic membrane was intact. It is highly probable that there had been a defect in the tympanic membrane, but that this had healed, leaving in the middle ear a bud of epithelium.

NOSE AND SINUSES

Willcox, Sir W.: Nasal Sinusitis as a Cause of Toxæmia. *Proc. Roy. Soc. Med.*, Lond., 1926, xix, Sect. Laryngol., 49.

Infection of the nasal sinuses is just as important in the causation of toxæmia as is dental sepsis. The diagnosis of acute nasal sinusitis is easily made. Chronic sinusitis is diagnosed on the basis of the history and the findings of expert examination of the nose including roentgenographic examination, transillumination, and rhinoscopic examination with puncture. From the author's experience with these cases he draws the following conclusions:

1. Nasal sinusitis is relatively common and should always be searched for in cases of toxæmia in which the cause is not apparent.

2. In cases of systemic disease which may be due to toxæmic conditions, a careful search should be made for sinusitis.

3. Nasal sinusitis is an important cause of toxæmia. It may be very far-reaching in its effects and may cause any of the many diverse pathological conditions which are now recognized as being sometimes due to dental sepsis.

4. Nasal sinusitis, particularly in the chronic form, often requires operative treatment. Adequate treatment is imperative since the condition is a focus of infection which, if left untreated, will speedily give rise to systemic disease affecting other parts of the body.

5. In the treatment of nasal sinusitis it should always be remembered that the case is a case of toxæmia, usually streptococcal, in which the focus of infection is in the sinuses. Every case is therefore a problem in immunity.

In the discussion of this report it was brought out that only chronic cases should be operated upon;

that asthma is often caused by nasal sinusitis; and that nasal sinusitis promotes a chronic toxic state by causing bronchiectasis. There was some disagreement as to the relative value of the X-ray and transillumination in the diagnosis, but it was generally agreed that neither is absolutely decisive.

MANFORD R. WALTZ, M.D.

McMahon, B. J.: The Pathology of Spheno-Ethmoidal Sinusitis. *Arch. Otolaryngol.*, 1926, iv, 310.

The author reviews seventy cases of spheno-ethmoidal sinusitis from the pathologist's point of view in order to determine whether or not there is any relation between the symptoms and the microscopic changes and between the microscopic changes and the end-results of operation on these sinuses. In every case the turbinate and cell walls removed at operation were fixed, embedded, sectioned, stained, and studied. From these studies the following conclusions are drawn:

1. In chronic hyperplastic spheno-ethmoidal sinusitis the microscopic examination shows thickening, sloughing, polypoid degeneration and metaplasia of the epithelium; thickening of the basement membrane; œdema, round-cell infiltration, dilatation or compression of the glands, and thickening of the blood vessel walls in the tunica propria, thickening of the periosteum; and osteoblastic activity, osteoclastic activity, fibrosis, hyperostosis, osteomalacia, and necrosis in the bones.

2. The symptoms which may be associated with these microscopic changes include headache, an anteronasal discharge, a posteronasal discharge, asthma, arthritis, failure of vision, impairment of hearing, and herpes of the second division of the fifth nerve.

3. There is no direct interrelation between the microscopic findings, the symptoms, and the results of operation.

4. The incidence of spheno-ethmoidal sinusitis is much greater among women than among men.

5. The percentage of good results is much higher among women than among men.

6. Chronic hyperplastic spheno-ethmoidal sinusitis is a distinct clinical entity in which the operative results are attended with improvement or complete recovery in a high percentage (74.3 per cent) of the cases.

MANFORD R. WALTZ, M.D.

MOUTH

Brown, G. V. I.: The Surgical Treatment of Cleft Palate. *J. Am. M. Ass.*, 1926, lxxxvii, 1379.

Thompson, J. E.: A Septal Flap in the Closure of Unilateral Clefts of the Palate. *J. Am. M. Ass.*, 1926, lxxxvii, 1384.

In the operation described by BROWN the velum and the posterior part of the hard palate are closed by a bone flap, and in the second step the anterior part of the palate fissure is corrected by any of the recognized methods in which a mucoperiosteal flap is used, such as the von Langenbeck procedure. Brown employs a bone flap when the palate fissure is complete and the bone is involved. In the cases of edentulous infants, incisions are made on both sides of the palate in an anteroposterior direction along the line of, and just inside, the alveolar ridge. When the teeth have erupted, they are made at just a sufficient distance from the linguogingival border to prevent injury to the tooth roots. A chisel is forced directly through the external hard surface of the fragmentary palate bone structures into the cancellous structures and then inclined toward the palatal fissure border.

Pressure inward and slightly upward causes the necessary fracture and permits both sides of the palate fissure border to be brought into immediate contact at a point between, and slightly anterior to what in a normal case would be the outline of the posterior border of the palate bones and also throughout the extent of the soft parts. The medial edges of each incision are sutured with pyoktanin catgut and pulled toward the midline. Gauze packs are inserted into the operative wounds for five days. To prevent blocking of the nasal passages by the packs, one or more nasal catheters are introduced.

Because of the unrestricted blood supply this method is associated with less danger of loss of tissue with a consequently imperfect result than other procedures. It gives a full length unstiffened velum and a posterior border of the palatal bones which favors the natural function of the attached muscles. The palate closure is completed from one and a half to two years before speech habits are fixed, no serious injury is done to the alveolar outline, the uninjured supporting bone framework tends to improve the outline of the lip, and the activity of both the posterior palatal and the pharyngeal muscles cooperating with the labial and facial muscles assists in progressive development toward more symmetrical facial and labial outlines, all factors of importance in the acquirement of good speech.

THOMPSON states that unilateral clefts of the palate should be operated upon early. The object of his operation is the restoration of the curve of the alveolar border and closure of the anterior part of the palate by means of a flap taken from the side of the septum. It results in union of the anterior ends of the alveolar processes and of a third or half of the front of the palate.

The septal flap is prepared first and the palatal flap next. An incision on the side of the septum is made horizontally from front to back along the ridge that separates the vertical and horizontal parts of the septum from each other. Its anterior end stops at the point in the groove which separates the alveolar ridge of the premaxilla from its palatal surface. Posteriorly the incision ends at the posterior border of the septum. The base of the flap is formed by the juncture of the septum and the mucoperiosteal layer of the hard palate.

This flap of mucous membrane is peeled from the septum with a periosteal elevator and retracted

toward the base. Behind, where the velum and hard palate blend, it is difficult to raise the flap because of the palatal aponeurosis, but after careful snipping away of the tissues the whole palate with the flap hangs free on its lateral and posterior attachments.

The palatal flap of mucoperiosteum is prepared somewhat similarly.

These flaps are now ready for approximation, but further preliminary steps are necessary before they can be sutured. The maxillæ, which are far apart, must be gently molded toward the midline and their tips denuded of mucous membrane. A silver wire suture is then passed through the maxillary processes to hold them together. The bones are penetrated at a considerable distance above the alveolar margins to avoid injury to the tooth sacs. The mattress sutures of the flaps are tied and the maxillæ pushed firmly together. The silver wire is then tightened.

Feedings are given immediately after the operation. The wire is removed after three or four weeks and the sutures of the flaps are removed after four or five weeks if they do not come away spontaneously. GEORGE R. MCAULIFF, M.D.

Figi, F. A.: Actinomycosis of the Tongue: Report of Twelve Cases. *Surg. Clin. N. Am.*, 1926, vi, 1343.

Primary actinomycosis of the tongue, while common in cattle and hogs, is rare in man, only thirty-seven cases having been previously reported. It has been observed most frequently in adult males engaged in agricultural pursuits.

In a typical case there is first noted a hard, painless, deeply situated nodule which in a few days comes to the surface, becomes tender and painful, softens, and ruptures. This process is repeated until the entire tongue and the floor of the mouth become indurated and thickened.

If the lesion is incised just prior to its rupture, sulphur bodies and actinomyces can usually be demonstrated in the pus. If a nodule is excised prior to softening, serial sections may be necessary to demonstrate actinomyces in the tissues. There is nothing else diagnostic in the microscopic picture.

The treatment consists in wide excision of early nodules and free drainage of those that have softened, together with radium irradiation of the glands and increasing doses of a saturated solution of potassium iodide up to 200 drops three times daily.

Eleven of the twelve patients whose cases are reported are well. The remaining one could not be traced, but should be well since the early nodule was widely excised.

PHARYNX

New, G. B., and Decker, W. J.: Pharyngeal Sinus with Cervical Pott's Disease; Report of Six Cases. *Surg. Clin. N. Am.*, 1926, vi, 1335.

Six patients with cervical Pott's disease have been examined at the Mayo Clinic during the last fifteen years. During the same period, twenty-four patients with tuberculosis of the cervical spine without pharyngeal abscess were examined.

There are four types of retropharyngeal abscess: (1) the acute, occurring laterally in the pharynx secondary to a nasopharyngeal or pharyngeal infection; (2) that affecting children, which is due to a pyogenic infection and usually associated with a similar infection in the cervical glands; (3) the tuberculous type, which is associated with tuberculosis of the cervical glands and is a breaking down of a tuberculous pharyngeal gland; and (4) the type secondary to tuberculosis of the cervical spine. The tuberculous abscess may involve the upper or lower cervical vertebræ. The symptoms vary with its situation.

Pharyngeal abscess associated with tuberculosis of the cervical spine occurred only in males. Four of the patients were young. One patient was a man aged 66 years and another a man aged 71 years. The symptoms extended over periods varying from six months to twenty-eight months.

Retropharyngeal abscess not associated with cervical Pott's disease occurs most commonly in young children. Of seventeen cases of retropharyngeal abscess examined which were not of tuberculous origin, eleven occurred in children less than 5 years of age. Of the thirty patients with cervical Pott's disease, twenty-two were adults, six of whom had pharyngeal sinuses, and eight were children without pharyngeal sinuses.

The part of the cervical spine involved was the first and second vertebræ in two cases, the second and third vertebræ in one case, the third and fourth vertebræ in one case, the fourth vertebra in one case, and the seventh vertebra in one case. In one case at the Mayo Clinic the seventh cervical vertebra was involved and the abscess ruptured into the œsophagus, causing an œsophageal as well as a cervical fistula.

NECK

Hudson, R. V.: The So-Called Branchiogenetic Carcinoma: Its Occupational Incidence and Origin. *Brit. J. Surg.*, 1926, xiv, 280.

Among cases of malignant disease there occasionally occur cases with a tumor of the neck in which, on careful examination, no discoverable focus of disease can be found. The tumor has the microscopic characters of a squamous carcinoma. In such cases the diagnosis rests between a primary carcinoma of branchiogenetic origin and a carcinoma secondary to a healed or undiscovered focus elsewhere in the body.

Hudson states that the prevalent opinion with regard to these apparently primary tumors of the neck may be briefly summarized as follows:

1. A solid malignant tumor of the neck showing the structure of a squamous-celled carcinoma is most probably secondary to a healed or undiscovered focus somewhere in the immediate vicinity.

2. A tumor originating in a vestigial remnant is rare, but occurs in two main types: (a) the branchiogenetic carcinoma; (b) the so-called endothelioma.

Ewing emphasizes the fact that branchiogenetic carcinoma is commonly cystic, but admits the possibility of a solid squamous-cell carcinoma of branchiogenetic origin.

The author reports in detail the histories of six cases of carcinomatous cervical tumor of obscure origin which occurred in miners or grooms.

Each of these tumors began as a small painless swelling just below and behind the angle of the mandible. The gradual increase in size of the swelling eventually compelled the patient to seek treatment for pain referred to the temporal region and the back of the neck. On examination the only positive clinical sign was the presence of an ovoid tumor regular in outline and firmly fixed to the skin and deep structures. The center of the growth lay just below and behind the angle of the jaw. In spite of rhinolaryngoscopy, no primary focus could be discovered. Microscopy revealed that the tumors were squamous in origin.

In all of these cases the cervical tumor arose at the site of the jugulodigastric gland. This gland is large, oval, and flattened, and about 2.5 cm. in length. It is situated upon the medial aspect of the great vein, a site which may be called the "critical point of the neck" since here, within the space of 2 or 3 cm., arises the vascular supply to the structures derived from the first five arches and to this point converge not only the greater part of their venous drainage, but also the lymphatic drainage.

The upper third of the sternomastoid laterally and the ascending ramus of the mandible medially tend to limit the spread of these tumors and favor the ovoid form which they maintain until a late stage in their development.

In only one case was an autopsy possible. In this instance the primary focus was in the pyriform fossa.

The fact that all of the tumors described occurred in miners or men who had worked with horses all their lives suggests that in occupations in which a certain anatomical tract is frequently subjected to trauma, such a site must be regarded as a possible portal of entry for a common causal agent.

With regard to treatment it may be stated that in the great majority of recorded cases surgical removal was disappointing; recurrence was generally rapid and fatal. The poor results are attributed by the author to the difficulty of early diagnosis due to the site of the tumor, and the presence of a primary focus acting as a neoplastic cell depot.

In the cases reviewed, operation was not considered. The patients were all hopelessly inoperable at the time they were first seen. The method chosen was radium irradiation. One of the patients is still alive and well, and in another the original tumor-bearing area was apparently free from growth at the time of death.

The diagnosis of these tumors rests principally upon their anatomical site and their consistency. The solid tumors that may occur at the same site are the primary endotheliomata of lymph glands of the single type and endotheliomata of branchiogenetic origin. Microscopic sections only will prove the diagnosis. The solid tumors occurring near the site, in addition to the tumors mentioned, are the endotheliomata and especially carcinomata beginning in the lower pole of the parotid gland, but these growths are always at a higher level, and more superficial and spread in the parotid substance and cheek as well as the post-ramal recess. Tumors of the carotid body are situated at a lower level and tend to be globular and early irregular in outline; their histological picture is definite.

An early diagnosis will always be difficult, but the presence of a unilateral painless tumor at the site of the jugulodigastric gland in an elderly man should be regarded with suspicion.

JACOB S. GROVE, M.D.

Menne, F. B., Joyce, T. M., and von Hungen, A. P.: Thyroid Disturbances: A Clinicopathological Study of 300 Instances. *Arch. Surg.*, 1926, xiii, 329.

The authors suggest the following classification of thyroid conditions according to their gross characteristics and the salient microscopic findings:

PATHOLOGICAL CLASSIFICATION OF THE NORMAL OR ENLARGED GLAND WITH OR WITHOUT INCREASED OR DECREASED ACTIVITY

1. Diffuse parenchymatous hyperplasia:
   - A. Gross examination: compact, ischæmic, grayish-white, and colloid-free.
   - B. Microscopic examination: (a) hyperplasia and hypertrophy of epithelium; (b) peripheral or general vacuolization of colloid; (c) dilatation of lymph channels and engorgement of blood vessels; (d) variable increase in the supporting stroma with or without round cell infiltration.
2. Diffuse adenomatous hyperplasia:
   - A. Gross examination: diffuse reddish-brown gland without noticeable nodularity or accentuation of lobular markings; variable amount of colloid.
   - B. Microscopic examination: (a) focal changes similar to those in Group 1; (b) normal or colloid stretched alveoli; (c) focal hyperplasia and hypertrophy of epithelium; (d) interalveolar hillocks or intra-alveolar papillomatous projections; (e) focal round-cell collections of pseudo-lymph nodes; (f) focal increased vascularity and dilated lymph channels.
3. Nodular adenomatous hyperplasia:
   - A. Gross examination: variable nodular accentuation of the lobular markings with or without excessive colloid storage, cystic degeneration, hæmorrhage, scarring, or lime salt deposit. The color usually varies with the regressive changes.
   - B. Microscopic examination: (a) focal cytological changes similar to those of Groups 1 and 2;

compensatory; (b) characteristic retrogressive changes; (c) areas of adenomatosis.

4. Solitary adenoma:
   A. Gross examination: variable in size; circumscribed; solitary or multiple; grayish-white to dark reddish-brown; solid, cystic, or colloid-filled; regressive changes may be present.
   B. Microscopic examination: (a) all stages of fetal types of alveoli; (b) peripheral pseudocapsule formation with round cell infiltration and compressed alveoli; (c) focal neighboring areas in adjoining parenchyma; (d) neighboring areas of adenomatosis.
5. Neoplasms.
6. Inflammation: (a) pyogenic; (b) infectious granuloma.

Of the 300 cases of thyroid disturbances reviewed, thirty-eight (12.6 per cent) belonged to Group 1, diffuse parenchymatous hyperplasia; 108 (35 per cent) to Group 2, diffuse adenomatous hyperplasia; 102 to Group 3, nodular adenomatous hyperplasia; forty-one to Group 4, solitary adenoma with hyperplasia; seven to Group 5, neoplasm; and four to Group 6, inflammatory processes.

The following conclusions are drawn:

1. A simple clinicopathological study is desirable in all thyroid diseases.

2. The incidence of so-called exophthalmic goiter (diffuse parenchymatous hyperplasia) in relation to other types is of importance.

3. Pregnancy is a factor precipitating thyroid unbalance.

4. The pulse pressure in thyroid disturbances is usually high; it is highest in the parenchymatous hyperplasia group. It is out of proportion to the ordinary changes in the blood vessels occurring in early and middle life.

5. The basal metabolic rate is a valuable indicator of thyroid unbalance and should be determined after operative procedures as well as before.

6. More experimentation is desirable to determine the significance of adenomatosis, solitary adenoma, and other pathological changes as well as the clinical effect of the amount of the gland removed.

JOHN J. MALONEY, M.D.

Keith, W. D.: Goiter from the Standpoint of Prevention. *Canadian M. Ass. J.*, 1926, xvi, 1171.
Gordon, A. H.: Goiter: Its Medical Aspect. *Canadian M. Ass. J.*, 1926, xvi, 1176.
McGuffin, W. H.: Goiter: From a Radiologist's Viewpoint. *Canadian M. Ass. J.*, 1926, xvi, 1182.
Fahrni, G. S.: Goiter: Its Surgical Treatment. *Canadian M. Ass. J.*, 1926, xvi, 1188.

KEITH reports that in Indian villages on the coast the inhabitants of which subsist to a large extent on salmon, salmon eggs, and seaweed, goiter is practically unknown. An Alberta survey, however, demonstrated that Indians are as subject to goiter as any other race. Further investigation proved that the incidence of goiter was increased where the drinking water was of a turbid or murky character. These waters are not iodine-free, but may contain iodine in an unassimilable form or some other organic or inorganic matter which has a definite influence upon the growth of the goiter.

For the prevention of goiter, Keith recommends the use of such food as salmon and other sea foods rich in iodine. The drug should be given in a goiter district for both prevention and cure, but its general use in the cases of school children should be carefully regulated in order that it may not cause toxic symptoms.

GORDON describes simple adenoma and cystic adenoma and points out that, in the latter, pressure on the trachea may cause symptoms of a confusing nature and even death from asphyxia. Acute thyroiditis may occur in the course of any of the infectious diseases and in certain cases of acute reactions due to infected tonsils.

Toxic goiter must be differentiated from simple goiter and from non-goitrous conditions. Tachycardia, tremor, and loss of weight are of much importance. In order properly to evaluate the basal metabolism, it must be remembered that the rate may be increased by many factors. Constant readings of + 10 or over may be considered as positive. Hyperthyroidism may be due to toxic adenoma, iodine hyperthyroidism, or exophthalmic goiter. The first type appears in persons of middle age in whom a thyroid tumor has existed for a number of years. The usual evidence is present but there is no exophthalmos.

Iodine hyperthyroidism may occur from self therapy or improper medication in cases of simple goiter. In exophthalmic goiter there is probably some other toxic element at work in addition to thyroxin as this condition cannot be produced by the administration of thyroxin or iodine. Myocardial damage in the chronic case may be due to the effect of toxins as well as to overactivity. In the treatment, iodine in proper doses is of value, even though its effects are temporary. Surgery offers the best hope of complete relief.

MCGUFFIN classifies goiter as:

1. Physiological goiter due to iodine deficiency.
2. Pathological goiter without hyperthyroidism. This includes diffuse colloid, parenchymatous, adenomatous, and cystic goiter.
3. Pathological goiter with hyperthyroidism, including toxic adenomata, and exophthalmic goiter.

With the exception of the last, these types are progressive from one to the other in the order given. Exophthalmic goiter shows hyperplasia of the epithelial elements lining the acini. In the treatment, mental and physical rest are important. Radium is preferred for the more serious cases because it is portable. The dosage is determined by the degree of toxicity and the basal metabolic rate. One hundred milligram hours are given over five areas for each 10 per cent increase in the basal metabolic rate. Treatments are repeated on each of five successive days and over each side of the neck every week for four weeks. The improvement is gradual.

The pulse rate falls, the nervousness ceases, the metabolic rate is diminished, and the patient begins to gain weight.

The author cites the advantages of X-ray and radium therapy and answers the objections advanced by the surgeon. He denies that the X-ray injures the parathyroid glands or produces adhesions about the gland. Most of the cases which do not respond to X-ray or radium irradiation have not been given sufficient treatment. Because of the number of surgical failures and the degree of operative risk, the author prefers X-ray or radium treatment to surgery.

FAHRNI states that although hyperthyroidism constitutes the large majority of surgical goiters, other forms, such as large colloid, cystic, and nodular goiters causing pressure, demand surgery. Adenomata have a tendency to become toxic in later years. These cases coming to operation late in toxicity constitute some of the most difficult cases. Many cases of colloid and adenomatous goiter are associated with a lack of energy, extreme fatigue, and mental irritability. These have been described as cases of "dysthyroidism," and are readily cured by removal of the gland.

The author reviews a series of 320 thyroidectomies. The condition for which the operation was done was exophthalmic goiter or primary hyperthyroidism in 55.93 per cent, toxic adenoma in 30 per cent, large adenomata in 6.25 per cent, large colloid nodular goiter in 3.75 per cent, cystic goiter (usually large and often associated with adenoma) in 3.75 per cent, and carcinoma in 0.31 per cent. Eight operations were done for recurrence following thyroidectomy performed from one to twelve years previously.

WILLIAM J. PICKETT, M.D.

Mayhew, J. M.: The Basal Metabolism and the Blood Chemistry. *Nebraska State M. J.*, 1926, xi, 409.

Emerson, C.: The Pathology of Goiter. *Nebraska State M. J.*, 1926, xi, 411.

Bliss, R. W.: The Medical Management of Goiter. *Nebraska State M. J.*, 1926, xi, 416.

Rowe, E. W.: The X-Ray Treatment of Goiter. *Nebraska State M. J.*, 1926, xi, 419.

MAYHEW cautions against relying entirely upon the basal metabolic rate in the diagnosis and treatment of thyroid conditions. He believes that the following three tests should be made:

1. The basal metabolism test plus clinical observation. In the absence of fever, acromegaly, leukæmia, and severe anæmia, an increased basal metabolic rate is strongly suggestive of hyperthyroidism.

2. The glucose tolerance test. In toxic thyroid conditions the return of the blood sugar to the normal is delayed from one to two hours.

3. The serological test evolved by Kottman, which is based on colloid chemistry. The technique of this test is described.

EMERSON states that the cause of dysfunction and associated anatomical changes in the thyroid gland is generally conceded to be an excitation due to a deficiency of iodine. Under this excitation, hypertrophy and then hyperplasia result. If the excitation is severe, exophthalmic goiter is produced. Extreme excitation induces atrophy and fibrosis with resulting myxœdema. Long-continued mild excitation gives rise to simple or toxic adenoma. If the excitation ceases, the histological picture returns toward the normal but evidence of the previous changes persists. This is the simple non-toxic colloid goiter.

Marine and Lenhardt have proved that a deficiency of iodine induces over-activity of the thyroid, but many clinical observations have demonstrated that the injudicious administration of iodine has stimulated over-activity.

The author presents a pathological classification of goiter according to the etiology.

BLISS discusses the medical management of goiter and draws the following conclusions:

Adolescent colloid goiter is prevented or greatly benefited by the administration of iodine, but the treatment of a community *en masse* with iodine is dangerous.

Colloid goiters in adults are benefited by iodine, but the patient requires careful supervision.

Benign adenomata are harmed by iodine and should be removed before they become toxic.

Exophthalmic goiter requires both medical and surgical treatment.

Digitalis is indicated only in auricular fibrillation or cardiac failure.

As infection may play a rôle in goiter, the eradication of all known foci is indicated.

ROWE states that clinical observations and records of the basal metabolism rate indicate that properly selected cases of goiter are as rationally treated with the roentgen ray as by surgery.

The general management of the patient is as important in roentgen-ray treatment as in other modes of therapy.

Cases of adenoma with toxicity yield more readily to surgery, but often show excellent results from roentgen-ray treatment.

Exophthalmic goiter shows the best results of all types.

In roentgen-ray treatment there is no mortality, no fear of the treatment, and no scar.

The treatment of toxic goiter is major roentgen therapy and demands skill.

The frequent check of the metabolimeter is an aid in diagnosis and treatment.

J. FRANK DOUGHTY, M.D.

Hotz, G.: The Operative Treatment of Basedow's Disease (Zur operativen Behandlung des Basedow). *Deutsche med. Wchnschr.*, 1926, lii, 604.

The more goitrous tissue that can be removed in Basedow's disease the more favorable is the result. However, extensive reduction is usually very dangerous. An important advance in the treatment of the condition is the prior determination of the basal

rate of oxygen exchange. In Basedow's disease this is very greatly increased. On the basis of the findings of this test the author has often operated in from two to four stages, first ligating the vessels and later resecting one or both halves of the thyroid gland. In this way the dangers may be materially lessened. Often the result is good after simple ligation of the vessels.

After a time, often after years, the complaints return following the establishment of the collateral blood circulation and further resection must be performed. The disadvantage of this method of treatment consists in its long duration and the difficulties attendant upon the second operation.

A further advance in the surgical treatment of Basedow's disease was brought about by the use of gynergen. This preparation is supposed to have an effect antagonistic to that of thyroxin. The pulse rate can be easily lowered by it. Permanent improvement of the symptoms following the use of gynergen alone was not observed.

The favorable effect of large doses of iodine on the acute phase of Basedow's disease is a very interesting and unexpected advance in the operative therapy. Following large doses of iodine (from 10 to 30 drops of Lugol's solution daily) the basal metabolism is reduced by 25 per cent. However this improvement is not permanent. The optimum effect is obtained in eight days. The basal metabolic rate then rises again. The iodine should therefore be given several days before the operation and particularly during the first days of the postoperative treatment. After eight days it should be discontinued. BRAUN (Z).

Korenchevsky, V.: The Influence of the Removal of the Thyroid, Parathyroid, and Sexual Glands and of Thyroid Feeding upon the Regulation of the Body Temperature of Rabbits. *J. Path. & Bacteriol.*, 1926, xxix, 461.

The cooling of normal young rabbits produces a more pronounced fall in the body temperature than the cooling of normal adult rabbits. After thyroidectomy, cooling causes a much more pronounced decrease and warming causes a much less pronounced increase in the body temperature than is observed in the normal animal before removal of the thyroid. Some adult thyroidectomized rabbits may die after a degree of cooling which normal rabbits are able to resist. The cooling of young thyroidectomized rabbits is lethal under conditions which, in the same rabbits before the operation, produced only a temporary fall of body temperature.

Parathyroidectomy does not change in a marked degree the response of normal or thyroidectomized rabbits to cooling or warming. In eleven experiments the influence of castration on the response seemed to be similar to that of thyroidectomy, but the changes produced were much less marked. The cooling of two rabbits in which both the sexual glands and the thyroid gland had been removed was followed by a lethal fall of the body temperature, a fact suggesting an influence of the sexual glands on the regulation of body temperature similar to that of the thyroid gland. Rabbits in which the thyroid, parathyroid, and sexual glands were removed simultaneously responded to cooling or warming in the same way as thyroidectomized animals.

The response of thyroidectomized rabbits to cooling or warming may be restored to normal by thyroid feeding. After long and excessive thyroid feeding, warming may even be followed by a lethal overheating with a rise in the body temperature to 43.5 degrees C. This effect of thyroid feeding upon thyroidectomized rabbits is produced only after about a week of thyroid feeding and does not disappear until several weeks after the cessation of the thyroid feeding. The resistance to cooling disappears before the resistance to warming.

In conclusion the author states that as the thyroid gland plays an important part in the regulation of the body temperature, its condition must always be taken into account in considering the resistance of different individuals to cold or heat and cases of disease accompanied by fever.

STANLEY J. SEEGER, M.D.

SURGERY OF THE NERVOUS SYSTEM

BRAIN AND ITS COVERINGS; CRANIAL NERVES

Demel: Living Dogs Whose Skulls Have Been Subjected to Roentgen Irradiation (Lebende Hunde deren Hirnschaedel mit Roentgenstrahlen bestrahlt wurden). *Zentralbl. f. Chir.*, 1926, liii, 2155.

Having demonstrated, during the past year, dogs which, following repeated irradiation of the skull, showed disturbances of growth, an ataxic gait, and atrophy of the eye-grounds, the author describes in this article the histological findings in the brain and eye-grounds of these dogs.

The irradiated brain showed a general decrease in size and atrophy of the right half, the side to which the irradiation was directed. The phylogenetically older portions of the brain withstood the irradiation better than the younger parts.

Histological examination showed a reduction in the pyramidal area and of the corpus restiforme in the medulla oblongata and a reduction in the numbers of fibers in other regions. The cortex showed a disturbance of layer formation, the disappearance of cells, and a vacuolizing, degenerative affection of the ganglion cells. There were no inflammatory or reactive processes in the blood vessels. Marked changes were found in the retina, which had in places almost disappeared. The choroid and optic nerve were apparently normal.

The histological findings explained the changes previously noted in the living animals.

JANSSEN (Z).

Sargent, P.: Types of Cerebral Tumors. *Brit. M. J.*, 1926, ii, 628.

Souttar, H. S.: A New Form of Craniotome for Opening the Skull. *Brit. M. J.*, 1926, ii, 630.

Bertwistle, A. P.: Localization by the X-Rays. *Brit. M. J.*, 1926, ii, 631.

In his discussion of the types of cerebral tumors, SARGENT calls attention to the pertinency, even today, of the remark made by Horsley twenty years ago with regard to so-called expectant treatment of intracranial tumors. Horsley said, "Considering that, in the absence of any active surgical treatment, the only thing to be expected is death, the term 'expectant treatment' has always to my mind carried with it its own condemnation for inhumanity."

Sargent brings out the fact that advances in surgical technique have robbed intracranial surgery of some of its terrors, and that in cases of cerebral tumors surgery offers the only hope of either a cure or alleviation of the symptoms. Tumors can now be more definitely localized, and even their nature can be predicted to some extent before the operation is performed.

Gliomata have a progressive and infiltrating growth but never metastasize or spread beyond the brain. If pituitary and cerebellopontine tumors are excepted, they constitute nearly 80 per cent of intracranial growths. They may be almost completely cystic and so degenerated that little, if any, recognizable tumor tissue is left, or firm, solid, and circumscribed but not encapsulated. For the more common rapidly growing and infiltrating type only palliative measures are possible.

In some cases Sargent has employed from 50 to 100 mgm. of radium for twenty-four hours. About 25 per cent of patients with cerebral gliomata die shortly after operation and about 50 per cent die within eight months after surgical treatment. About 25 per cent make good recoveries, surviving for several years and being able, during that time, to earn their own living.

In cases of cerebellar gliomata the results are better. The operative mortality is less than half that of cerebral gliomata. Twenty-eight per cent of the patients are alive and well on an average of three years after the operation, and the average survival of the remainder is thirteen months. Sargent cites the cases of three patients who are clinically cured.

Endotheliomata are benign, slowly growing encapsulated tumors arising from the cells of the arachnoid tufts. They do not actually invade the brain, but form depressions in it. Complete removal requires the removal of the dura overlying the tumor and this is sometimes fraught with danger and difficulty on account of the presence or proximity of the large venous sinuses. Endotheliomata are relatively rare. Of seventy-five cases in which they were completely removed, a good recovery resulted in 50 per cent. Patients who recover may be divided into two groups, those restored to their normal lives and occupations with no neurological defect (22 per cent), and those with a neurological defect such as palsy, convulsions, etc. (38 per cent). The operative mortality is high.

The majority (90 per cent) of cerebellopontine tumors are neurofibromata. As a rule these growths are firm, solid, and encapsulated, but they may be soft and even cystic. For years, the only complaints may be deafness and head noises. Bárány tests are positive in the early stages. Often the corneal reflex is lost or diminished. In all cases of nerve deafness the corneal reflex should be determined and the Bárány test carried out. A cerebellopontine tumor eventually compresses the aqueduct. When ventricular distention occurs with the classical signs of increased intracranial pressure, operation becomes more dangerous and difficult and the outlook is grave. On account of the proximity of the tumor to the medulla and its blood vessels its excision *en*

masse is hazardous. It should be removed by intracapsular enucleation in fragments and by suction.

Pituitary tumors are divided into two groups: (1) pituitary tumors proper, most of which are adenomata, arising in the sella turcica and invading the cranial cavity, and (2) suprapituitary tumors, arising above or in close relation to the sella. Judged from the point of view of visual improvement—and in most cases there are visual disturbances—the gain from operation greatly outweighs the operative risk. It is certain that as these cases come to operation earlier and before the visual pathways have been severely damaged, better results will be obtained. Decompression will render the patient more comfortable as it affords relief from headache and often results in improvement in the mentality.

In conclusion Sargent says that the outlook for further improvement in cases of intracranial tumors depends to some extent on the further improvement of surgical technique, but even more upon earlier diagnosis and accurate localization. The onset of the classical signs—headache, vomiting, and papilledema—indicates the beginning of the terminal stage of the condition. It is of vital importance that surgical intervention take place before this stage is reached. A careful neurological examination should be made in every case of disturbance of cerebral function, especially when it is persistent or progressive. In many cases there will be a history of some slight disorder which has been present for months or years.

Souttar describes a new form of craniotome for opening the skull with the use of which his average time for turning down a large bone flap is between two and two and a half minutes. The appliance consists of a stud to be fixed in a trephine opening in the center of the bone flap, which carries an arm or lever that can be swung around in a circle and upon which the cutting instrument can be fixed at any desired distance from the center. This instrument cuts through the bone very rapidly and with little exertion on the part of the operator. It carries a guard to prevent injury of the dura. Souttar uses also a specially devised brace and bit for trephining.

Bertwistle describes a method of localization of various parts of the brain by means of the X-ray. In this procedure he uses an apparatus made of watch-spring steel which consists essentially of a base line extending from the glabella to the external occipital protuberance, upon which are erected at right angles, at accurately designated distances, a number of upright members. This apparatus is placed upon the head, a lateral roentgenogram is made, and, by means of measurements and calculations, the various gyri can be localized and projected upon the skull cap. In this manner an exploration can be made exactly over any desired area. For measuring these distances, Bertwistle uses an inexpensive celluloid instrument which he calls a "gyrometer." The method is of value particularly in cases of depressed fracture, but is helpful also in cases of brain tumors. Gilbert C. Anderson, M.D.

Heymann, E.: Clinical Experiences with the Development and Removability of Tumors at the Cerebellopontine Angle on the Basis of Twenty-Two Observations (Klinische Erfahrungen ueber die Entwickelung und Entfernbarkeit der Kleinhirn-Brueckenwinkelgeschwuelste auf Grund von 22 Beobachtungen). *Beitr. z. klin. Chir.*, 1926, cxxxvi, 385.

The author designates tumors at the cerebellopontine angle as tumors of the acoustic nerve. He discusses the diagnosis and prognosis of these tumors on the basis of twenty-two cases. The unilateral acusticus disturbance and the simultaneous failure of the reflexes of the cornea are absolutely definite focal symptoms. In addition, there may also be signs in the adjacent areas. Choked disk and headache in the opposite frontal region are common. The neurological and the otological findings render other methods of diagnosis unnecessary.

The operative procedure is described in detail. Of the author's patients, 43.7 per cent remained alive for some time after the operation. When blindness has once developed, operation is useless.

Koch (Z).

Eagleton, W. P.: Otitic Meningitis. *J. Am. M. Ass.*, 1926, lxxxvii, 1544.

From the standpoint of operation, cases of suppurative meningitis at an early stage may be divided according to the region of protective reaction as follows:

1. Posterior fossa cases:
   - A. Labyrinth cases: (a) from infection through the internal auditory meatus; (b) from infection by way of the ductus endolymphaticus; (c) from caries of the posterior semicircular canal.
   - B. Cases from caries of the petrous pyramid without labyrinthitis—Trautman's triangle, etc.
   - C. Cases secondary to thrombophlebitis of the lateral sinus.
2. Middle fossa cases:
   - A. Secondary to caries of the tegmen, apical cells, superior semicircular canal.
   - B. From thrombophlebitis of the small vein.
   - C. Associated with osteomyelitis of the squamous.

In any of these the operative discovery of the causative pathological lesion will determine the area of the cerebrospinal fluid system to be drained.

In septic meningitis, operation should be done while the inflammation is limited to an area adjacent to the primary focus of infection or an adjoining basal cisterna. It is during the period of apparent quiescence, when there are slight signs of meningeal involvement, that surgery offers a fair prospect of recovery.

There is no doubt that the body frequently succeeds in spontaneously overcoming the infection in many cases, especially if the increased intracranial pressure is relieved by repeated lumbar punctures. In two of the author's cases in which death occurred

from other causes, autopsy showed spontaneous recovery from the meningitis with localized adhesions.

The ventricles are not invaded until the terminal stage. Surgery is then useless, although in the near future irrigation (of the ventricles) may save life.

There are three distinct pathological conditions in meningitis which are amenable to surgery during an early stage: (1) an excess of infective fluid with or without exudate within the meshes of the arachnoid, (2) necrosis of the cerebral cortex with excessive exudate, and (3) meningeal exudate into the subdural space secondary to a brain abscess or a complicating sphenoid or accessory sinus suppuration.

The first requires simply evacuation through a small dural opening. In the second a large dural opening is necessary. This is apt to be followed by brain herniation. The relief of the third calls for extensive incision of the dura without injury to the underlying pia arachnoid.

There is no surgical panacea for terminal septic leptomeningitis. No one surgical procedure is uniformly successful even in the early stage of the condition. The surgeon must diagnose the exact site while the infection is limited, and then surgically attack it. The manner in which this is done must depend upon the conditions in the particular case.

The principles of surgical treatment are:

1. The eradication of the primary focus of infection.

2. Evacuation of the infected fluid in the basal cisterna by small dural openings, associated with drainage by punctures in the occipito-atloid and lumbar regions or, in cerebral necrosis or exudate in the subdural space, evacuation by a large dural incision.

3. Ligation of the common or internal carotid to place the parts at rest.

The after-treatment should include repeated lumbar punctures associated with transfusions from an immunized donor to increase the immunity of the cerebrospinal fluid by osmosis of the immunizing contents of the blood into the cerebrospinal fluid system. TRACY J. PUTNAM, M.D.

Brunner, H.: Plastic Operations for Facial Paralysis (Ueberplastische Operationen bei Facialislaehmung). *Arch. f. klin. Chir.*, 1926, cxl, 85.

The results of the various types of neuroplasty and suspension methods with living as well as with dead material in the treatment of facial paralysis are quite uncertain. More chances of success are offered by the muscle plastics in which strips of tissue of trigeminal innervation are used. Muscular strips are most frequently taken from the masseter according to the suggestion of Lexer and Jianu. For plastics on the eyelid and those on the angle of the mouth the temporal muscle has been employed (Lexer, Jianu, Krause).

The operation is usually carried out through an incision on the cheek, i.e., through the area of distribution of the facial nerve. The author describes a method which makes it possible to avoid the rather disfiguring cut on the outer side of the face and gives the facial nerve an opportunity to recover, which it will not do if its branches have been severed.

The incision is made upward from the zygomatic arch. A strip from the anterior border of the temporal muscle is then loosened from its insertion and carried down over the zygomatic arch, which may be notched for it, through a bluntly bored tunnel under the skin of the cheek to the corner of the mouth, where it is sutured through a very small opening.

In other cases the author uses the masseter as suggested by Lexer. He approaches it, not through a lateral facial incision, but through the mouth. The medial border of the muscle is exposed and a strip reflected and sutured into the region of the paralyzed orbicularis oris.

In all cases the results have been satisfactory. In no patient has infection occurred. Five cases are reported. RUGE (Z).

PERIPHERAL NERVES

Hertzler, A. E.: The Nature and Treatment of Sciatica. *Am. J. Surg.*, 1926, n.s. i, 200.

Hertzler defines sciatica as a painful affection of the sciatic nerve due to conditions within the nerve itself, produced by causes unknown or by irritating lesions adjacent to the nerve, and persisting after the subsidence of those conditions. Pain in the sciatic nerve which is the result of some demonstrable organic disease should not be classified as sciatica.

The cause of sciatica is unknown. The condition may be due to a local irritation such as myositis or a sacro-iliac affection or it may have some unknown toxic source. It is commonly preceded by pain in the sacrolumbar region after unusual exertion or exposure resulting in either a myositis or synovitis of the sacro-iliac joint. When it begins as a myositis, deep injections into the quadratus lumborum and the use of local anæsthetics, heat, massage, and antirheumatics often relieve the muscle pains. When the initial pain is felt in the sacro-iliac region there is probably a sacro-iliac synovitis with secondary involvement of the sciatic nerve. When the onset is insidious the cause may be a hidden focus of infection.

The symptoms usually begin gradually but may come on abruptly. The pain is felt most commonly where the nerve crosses the head of the femur, in the outer border of the popliteal space, or along the peroneal or posterior tibial nerves. It is intensified by exposure to cold, by movement, and by pressure. It is often most severe in the early morning hours. The entire nerve may be tender to palpation or no tender points may be found. The classical test is Lasegue's test with the patient lying, sitting, or standing. Minor signs of the condition are shrinkage of the Achilles tendon, flabbiness of the thigh musculature, and relative coolness of the affected side.

The differential diagnosis calls for a complete examination of the patient, including X-ray examination. The most important conditions to rule out are pressure on the nerve by tumors, inflammatory lesions such as tuberculosis and infectious arthritis, and affections of the cauda equina and lower cord.

The treatment of the pain in the back includes rest in bed and counterirritation with chemicals or wet or dry heat. The older practitioners gave large doses of belladonna, hyoscyamus, or potassium citrate. These are still regarded as of value, but more recently atophan (30 to 50 gr. per day) has been found almost a specific for the pain.

The remedies which have been used in the treatment of the sciatica are legion. They include operative and non-operative stretching of the nerve and the application of supporting apparatus to the pelvis, both of which are useless, and the injection into or around the joint of simple fluids or local anæsthetics, which sometimes gives good results. For many years the author has injected the nerve where it crosses the neck of the femur with 1 oz. of a 1 per cent solution of quinine urea hydrochloride. He has had no failures or recurrences with this treatment and only one unfavorable result, viz., infection requiring drainage. ALBERT S. CRAWFORD, M.D.

SYMPATHETIC NERVES

Isakowitz, H.: Injury of the Heart Muscle After Division of the Extracardial Nerves, and a Contribution on the Operative Treatment of Angina Pectoris (Herzmuskelschaedigung nach Durchtrennung extrakardialer Nerven; zugleich ein Beitrag zur operativen Behandlung der Angina pectoris). *Zentralbl. f. Herz- u. Gefaesskrankh.*, 1926, xviii, 105.

The theoretical basis of the operative treatment of angina pectoris is still so uncertain that every operation has the value of an experiment which may help to answer the numerous questions of an anatomical, physiological, and pathogenetic nature regarding the syndrome of angina pectoris.

The operative procedures on the vagus and cervical and thoracic sympathetics in man may be divided into two groups, those done for angina pectoris and those done for other conditions such as bronchial asthma, glaucoma, epilepsy, and Basedow's disease. Those of the second group are of interest since they show how the exclusion of the extracardial nerves can be tolerated by the normal heart.

Nothing regarding cardiac damage following these operations is to be found in the literature. However, the period of observation is still very short. On the other hand, the effects upon the heart of operations for angina pectoris are very unfavorable. In the seventy-five cases reviewed by the author, there were nineteen deaths and at least eleven of them appeared to be of cardiac origin.

In the author's opinion, operations for angina pectoris should be limited to cases in which internal medication is without avail and the condition is so precarious that a perilous measure is justified.

COLLEY (Z).

Kuemmell: The Causes of Failure in Operations for Asthma and Their Prevention (Die Ursache von Misserfolgen bei Asthmaoperationen und ihre Verhuetung). *Zentralbl. f. Chir.*, 1926, liii, 1278.

The chief purpose of Kuemmell's operation for asthma is the exclusion of the pathological hyperirritability of the bronchoconstrictors by severance of the nerve tracts leading to the central organ.

Braeucker's careful experimental work has clarified the anatomy of the vagus and sympathetic. It has shown that in the severance of the sympathetic as done by Kuemmell as well as in the deeper severance done below the level of the recurrent laryngeal nerve (the Kappis procedure), the lower pathways remain intact. Kuemmell severs the pulmonary branches at the point where the sympathetic fibers join those of the vagus. Thereby only the posterior bronchial rami are severed, the anterior bronchial rami being left intact.

The newer operation is performed anteriorly in the fourth intercostal space. The distended lung is pushed aside and the posterior bronchial rami are exposed and severed.

This operation has been performed twice by Kuemmell and once by Ludeck. In every instance it was successful.

In the discussion of this report, BRAEUCKER reviewed his experiments on animals. GLASS (Z).

Dittler, R., and Mueller, H. K.: Experiences with Regard to the Efficiency of the Vagus After Bilateral Extirpation of the Stellate Ganglion (Erfahrungen ueber die Wirksamkeit der Herzvagus nach doppelseitiger Extirpation des Ganglion stellatum). *Ztschr. f. d. ges. exper. Med.*, 1926, l, 166.

To determine whether there are any demonstrable changes in the effect of the vagus on the heart following bilateral extirpation of the stellate ganglion with subsequent nerve degeneration, the authors performed experiments on nerve-intact cats and cats in which the nerve had been operated upon. The length of time between the operation and the tests was at least eighteen days.

Threshold determinations as well as determinations of the time of development and the degree of the vagus effect upon marked stimulation of the nerve showed no definite variation in the behavior of the animals compared. Continuous stimulation of the vagus of definite strength gradually became less effective in both groups of animals. The transitory inhibition of vagus action following the intravenous administration of adrenalin solution was perceptibly weaker in the animals operated upon than in the controls and sometimes failed entirely. In such cases (three in ten) the adrenalin caused, after the first injection, a distinct increase instead of a decrease in the effectiveness of the vagus on the heart. STAHL (Z).

SURGERY OF THE CHEST

CHEST WALL AND BREAST

Swan, R. H. J., and Fry, H. J. B.: Tuberculosis of the Male Breast. *Brit. J. Surg.*, 1926, xiv, 234.

Tuberculosis of the male breast must be accounted a pathological curiosity. Not more than twelve cases have been recorded in the literature since the condition was first described nearly a century ago. In spite of the widespread prevalence of tuberculosis in its various forms, tuberculous infection of the breast, even in the female, is a rare condition.

The authors report the case of a man 42 years of age who complained of a swelling in the left breast which he had noticed for three months. During the last month there had been some retraction of the nipple. Twenty-four years previously the patient was operated upon for tuberculosis of the right hip joint, which was ankylosed.

Examination of the breast revealed a rounded, hard, somewhat nodular swelling about 2 in. in diameter which was fixed to the subjacent pectoral muscle. The nipple was slightly retracted. The skin of the areola and of the part immediately surrounding it was thickened and adherent to the mass, but there was no redness or œdema. Several small glands were palpable in the left axilla. A diagnosis of carcinoma of the breast was made. Radical removal of the breast was followed by complete recovery.

The specimen showed an abscess cavity immediately beneath the nipple in the fatty areolar tissue between the pectoralis major and the skin. On examination of the pus from the abscess a considerable number of tubercle bacilli were found.

Following the operation the patient was examined for other evidences of tuberculosis. Small hard nodules were found in the epididymis on both sides and one was discovered in the upper part of the right lobe of the prostate. In the absence of a history of venereal disease these were looked upon as old foci of tuberculous infection.

Tuberculosis of the breast is of two types, primary and secondary. The infection is considered to be primary when it occurs either directly through the skin of the breast or through the ducts of the nipple or is conveyed to the breast through the blood stream from a remote portal of entry. The type which is secondary to foci elsewhere is the commonest.

Various pathological types of tuberculous disease of the breast are described, namely: (1) acute miliary tuberculous mastitis; (2) the nodular type; (3) the sclerosing type; and (4) mastitis obliterans. Only the nodular type appears to occur in the male.

The condition can be recognized with certainty by the finding of tubercle bacilli in the tissues or in the pus of the abscesses, by their cultivation from the pus, or by animal inoculation in suspected cases.

The course of the disease is rapid in the male—not more than from three to six months—and shorter than in the female. This is evidently due to the fact that a small lump is recognized more easily in the rudimentary male breast and treatment is instituted earlier. In at least half of the cases the initial sign was a lump in the breast. Pain was present in only two cases. The site is usually the region of the nipple.

The condition must be differentiated from ordinary pyogenic mastitis and abscess, granulomata (actinomycosis, syphilis), simple and malignant tumors, and fat necrosis.

The prognosis appears to be uniformly good. Radical excision of the breast with the removal of all tissues involved and with or without clearing of the axilla seems to be the method of choice. Incision and drainage has also proved effective.

JACOB S. GROVE, M.D.

TRACHEA, LUNGS, AND PLEURA

Desjardins, A. U.: The Reaction of the Pleura and Lungs to Roentgen Rays. *Am. J. Roentgenol.*, 1926, xvi, 444.

Inflammatory reaction of the pleura and lungs to irradiation may occur in any case in which a sufficient dose of radium or roentgen rays has been directed to these structures. The symptoms of pleuropneumonitis may appear from two to four weeks after a course of roentgen-ray treatments. The chief symptoms are a cough, shortness of breath, and sometimes fever. In from one to three weeks their acute phase subsides as the remaining lung tissue adapts itself to the new functional requirements and as the more or less injured pulmonary parenchyma recovers its normal activity. The physical signs of pleuropneumonitis vary considerably, depending upon the degree, extent, and situation of the pleural or pulmonary tissue injuries. Râles and a pleural rub or pleural effusion may occur. Such physical signs diminish to a large extent but evidence of chronic pleuropneumonitis persists in the form of adhesive bands or more or less extensive adhesion of the pleural layers. If sufficient pulmonary tissue has been involved, a varying degree of functional impairment results, leading to compensatory emphysema of the remaining pulmonary parenchyma.

During the early phases the roentgenological signs may consist merely in the diffuse fog of inflammatory pleural thickening, with or without the still denser shadow of effusion, or they may simulate

fairly closely those of the focal type of pneumonia so commonly seen after influenza. Since the site of such infiltration must necessarily depend upon the conditions of the roentgen-ray treatment, the possibility that the roentgen rays may be responsible for the pleural and pulmonary disease process must include the coincidence of cause and effect in the same region.

The differential diagnosis between pleuropneumonitis following roentgenization and certain forms of pulmonary metastasis may be difficult. This distinction may require periodic observation. In certain cases the differentiation between the pleuropneumonitis due to roentgenization and lung abscess must be made; such a distinction is rarely difficult if the clinical features of pulmonary suppuration such as the abundant expectoration of pus and the irregular and rather high fever are borne in mind. It may be necessary also to distinguish a pleuropulmonary reaction to the roentgen rays from other inflammatory manifestations, but the correlation of the history and physical findings with the history of roentgen-ray exposure usually constitutes a satisfactory basis for the diagnosis.

Nearly all writers on the subject have conveyed the impression that pleuropneumonitis following irradiation of the thorax is related in some way to the use of roentgen rays of short wave length. While the voltage at which the rays are generated and the filter selected probably exert a certain limited influence on the process, the chief factor is quantitative and related to the duration of the exposure. In other words, inflammatory manifestations in the pleura and lungs are more likely to supervene the nearer the dosage approaches or passes beyond the limit of skin tolerance. In the author's experience pleuropulmonary reaction never follows the first course of roentgen-ray treatment and seldom follows the second, but on further treatment given with full dosage the occurrence of such manifestations is probable.

ŒSOPHAGUS AND MEDIASTINUM

Maydl, V.: A Case of Fatal Rupture of the Cardia in Dilatation for Cardiospasm with Stark's Sound (Ein Fall einer toedlichen Kardiaruptur bei einer Kardiospasmusdilatation mittels Starckscher Sonde). *Med. Klin.*, 1926, xxii, 408.

The author used Stark's method of dilating for cardiospasm in seven cases with very good results. The patients, who had been unable to swallow solid food for from two to twelve years, were completely cured. In three cases the dilatation decreased from 1 to 3 cm., the previously atonic œsophagus having re-acquired its peristalsis and tonus. The dilatations were all done on ambulatory patients. The pain felt in the breast at dilatation soon ceased.

The fatal rupture caused by the dilatation occurred in the case of a man 24 years of age who was suffering from œsophageal symptoms which had become so severe in the last four years that he was able to swallow only fluids. Numerous treatments with sounds and the administration of drugs to overcome the spasm were of no benefit. The roentgen-ray examination made with a contrast meal showed dilatation of the œsophagus with the formation of an isthmus. From this point only a very fine passage led into the stomach. Stagnation of the contents of the œsophagus lasted for six hours. By means of the œsophagoscope it was possible to see the spastically contracted mouth of the cardia at a depth of 43 cm. No trace of infiltration or ulcer was noted. A Starck sound was passed under X-ray control, and the dilator fitted into place in the passage, a procedure that did not require any particular exertion of force. No blood appeared on the sound. That night the patient suddenly collapsed. Death occurred eighteen hours after the dilatation.

Autopsy revealed a tear 1½ cm. long and ½ cm. wide in the mucosa and musculature of the posterior wall of the cardia directly below the diaphragm, and a diffuse peritonitis. GUSTAV (Z).

SURGERY OF THE ABDOMEN

ABDOMINAL WALL AND PERITONEUM

Ssokolovskij, M. P.: The Absorption of Bacteria from the Abdominal Cavity (Beitraege zur Frage ueber Resorption der Bakterien aus der Bauchhoehle). *Vestnik chirurgii i pograničnych oblastej*, 1925, iv, 20.

The author studied the conditions of resorption of bacteria from the abdominal cavity, especially the importance of the lymphatic system and omentum in this process, in a series of experiments on dogs. Various kinds of foreign bodies, such as dyes and hen's erythrocytes, and of bacteria, such as bacillus pyocyaneus and bacillus prodigiosus, were introduced into the abdominal cavity. The lymph from the thoracic duct was then studied.

The bacteria were found in the arterial and venous blood as well as in the lymph of the thoracic duct after from ten to fifteen minutes. Ligation of the thoracic duct prevented their entrance into the general circulation. Extirpation of the great omentum did not reduce the resistance of the body to infection and had no influence upon the resorption of bacteria and foreign bodies.

The most important conclusions to be drawn from these experiments are the following:

1. The resorption of bacteria and insoluble powders introduced into the abdominal cavity occurs by the lymphatics and not by the blood vessels.

2. The lymphatics have a marked capacity for resorption.

3. In resistance to an infection an important factor, in addition to the action of the great omentum and the peritoneal exudate, is the resorption of bacteria through the lymphatic vessels into the general circulation. The latter results in a mobilization of all of the protective powers of the body.

ALIPOV (Z).

Kirschner: The Treatment of Suppurative Diffuse Peritonitis (Die Behandlung der eitrigen, freien Bauchfellentzuendung). *50 Tag. d. deutsch. Ges. f. Chir.*, Berlin, 1926.

Kirschner first reviews briefly the various hypotheses advanced to explain deaths from suppurative diffuse peritonitis. According to one theory, the toxins cause paralysis of the vascular centers in the medulla oblongata. This supposition is open to the objection that the blood pressure falls only a short time before death. It is more probable that the toxic effect is exerted upon the capillaries of the abdominal cavity, as the result of which the portal circulation is injured, the blood accumulated in the abdominal cavity being transported no further. There is, as it were, a hæmorrhage into the abdominal cavity. However, even this theory is not supported by conclusive evidence. Therefore, for want of an explanation of the cause of death, reliance must be placed upon statistics to determine the proper method of treatment.

From a review of the cases treated at the Koenigsberg Clinic during the last thirty years and 10,000 case records which Kirschner collected by a questionnaire sent to twenty-three clinics and hospitals, it becomes apparent that the cause of peritonitis is a factor in the outcome of the condition. In the cases due to appendicitis, the mortality was 35.2 per cent, in those due to gastro-intestinal perforation it was 58.2 per cent, and in those following operation it was 100 per cent.

Moreover it appears that the resistance to the disease varies at different ages. Of 1,000 patients, 13.5 per cent died before the fifth year of age; 1.99 per cent in the fifteenth year; 10.1 per cent at the age of 50 years; 20.3 per cent at the age of 60 years; and 44.3 per cent at the age of 70 years.

The constitution also plays an important part in resistance to the condition. Of chief importance, however, is the time at which operation is done. The mortality of operations performed within the first twelve hours was only 24.9 per cent, while that of those done within from twelve to twenty-four hours was 32.4 per cent, that of those done after from twenty-four to forty-eight hours, 45.4 per cent; and that of those done after forty-eight hours, 66.6 per cent. Therefore every case of acute diffuse peritonitis which is operable should be operated upon as soon as possible. When the diagnosis is certain, the only exception to this rule are cases of peritonitis due to the gonococcus and the pneumococcus. The latter are rarely diagnosed before operation, the majority of cases coming to operation under the diagnosis of appendicitis.

The author emphasizes that, with the exception of Pels-Leusden, all of the surgeons to whom the questionnaire was sent were in agreement with regard to the fundamental principle of immediate operation.

In laparotomies, the chief essential is gentleness in the handling of the tissues. The incision in the abdominal wall is made directly above the suspected focus of disease or, when there is doubt, in the midline. It is made large enough to allow a good view of the disease focus and an easy approach to it. The main object of the interference is the certain removal of the source of infection in the simplest manner. If possible, a perforated organ should be extirpated. An exception to this rule, in the opinion of most surgeons, is the perforated gastric ulcer, for which more conservative operative methods for the elimination of the infection are advisable. The eventration of large sections of gut should be avoided as much as possible.

With regard to the manner of removing exudate found in the abdominal cavity—whether this should be removed by irrigation or by dry sponging—there is a difference of opinion. Thirteen of the clinics to which the questionnaire was sent were in favor of irrigation, fourteen in favor of sponging, and five reported that they sometimes follow one plan and sometimes the other. The statistics do not show any marked difference in the results. The author recommends irrigation with physiological salt solution for the removal of exudate which is uniformly distributed over the entire abdominal cavity, and sponging for the removal of that which is localized.

Kirschner is opposed to the introduction of drugs such as ether, camphorated oil, elmocid, pepsin-hydrochloric acid and rivanol, into the abdominal cavity. Only two of the surgeons questioned were in favor of it. The mechanical emptying of the gut at operation, which is so important in ileus, and the primary formation of an intestinal fistula should be omitted. Just as ineffective are attempts at drainage of the free abdominal cavity. If it is possible definitely to remove every focus of infection, the abdominal wound should be closed completely. If such thorough removal is impossible, only the locally circumscribed focus of infection should be drained. This can be walled off from the free abdominal cavity by tamponade. Investigations have shown that the drain is completely walled off after from twelve to twenty-four hours. The secretion that comes away thereafter is only the secretion from the drainage canal. Drainage of the cul-de-sac of Douglas, first suggested by Rehn, and the Rehn-Fowler low position of the pelvis, which have been recommended for this purpose, do not lead to the desired result. They should therefore be omitted and the patient placed in the horizontal position or the position that is most comfortable.

In the after-treatment, the general resistance, the tone of the blood vessels and capillaries, and the heart action should be strengthened. Camphor preparations, especially when used indiscriminately have not proved as beneficial as digitalis and suprarenin. Especially important is the use of morphine, the sovereign remedy for sparing the heart. Fluid should be administered by rectal and intravenous drip infusions in quantities sufficient to produce from 1 to 1½ liters of urine daily. Instead of sodium-chloride solution, normosal and glucose solution may be used. According to recent investigations it is advantageous to add insulin to improve the utilization of the sugar.

When vomiting occurs, periodical lavage of the stomach or drying of the stomach by means of a retention catheter through the nose and aspiration are of value. Of greatest importance is the stimulation of intestinal function. This evacuates the toxins accumulated in the intestine, removes the blood from the portal circulation, and, as was shown by the experiments of Usadel, acts against the stagnation of blood that occurs in the abdominal cavity. For this purpose, use may be made of rectal enemas and the parenteral and oral administration of cathartics.

Pituitrin and neohormonal have often proved of value. The local application of heat tends to stimulate intestinal activity. If it is impossible to induce bowel movements by this treatment, the enterostomy of Heidenhain comes up for consideration and, in desperate cases, multiple percutaneous punctures of the gut are justified.

By following these principles it has been possible to reduce the mortality in all cases at Koenigsberg from 87.5 to 30 per cent, that of peritonitis after appendicitis from 83.3 to 20.8 per cent, and that of peritonitis after perforations of the gastro-intestinal tract from 100 to 42 per cent.

In the discussion of this report, STAHNKE (Wuerzburg) discussed the relation of resorption from the abdominal cavity to the sympathetic nervous system. As the vagus and splanchnic nerves have an influence on the distribution of blood in the abdominal cavity and the permeability of the cells, they influence resorption. Stahnke demonstrated this influence in experiments on rabbits and dogs in which he first used potassium iodide and then the fluorescein test. After section of the splanchnic nerves there was an acceleration of the resorption of the exudate with lengthening of the total duration of the process, whereas after division of the vagi there was acceleration for the first few days, but then a retardation. Experiments with morphine showed a retardation in 50 per cent of the animals and an acceleration in the other 50 per cent. When peritonitis was induced artificially the resorption remained unchanged after section of the splanchnic nerves, whereas section of the vagi was followed by retardation.

A difference was evident in the reactions of the dog and rabbit. In the dog, resorption was accelerated after section of the vagi as well as after section of the splanchnic nerves, whereas in the rabbit it was accelerated only after section of the vagi. In peritonitis, retardation of resorption and marked retardation of excretion followed section of the splanchnics as well as section of the vagi. It is evident from these findings that in splanchnic anæsthesia resorption is accelerated in cases without peritonitis and retarded in cases with peritonitis.

VOGT (Tuebingen) discussed intravenous pituitrin-sodium chloride infusion in postoperative peritonitis. He stated that he had seen a very favorable effect from the intravenous injection of 500 gm. of normosal with four or five ampoules of pituitrin. This treatment stimulates intestinal activity and has a favorable effect on the vascular system. The pulse is retarded and the vasomotor paralysis disappears. The toxins present in the body are diluted. Diuresis is stimulated. Frequently one large infusion is sufficient, but occasionally must be repeated two or three times. Vogt obtained a cure with this treatment in 22 per cent of eighty-one cases of postoperative peritonitis. Koerte asked in what manner it was determined that these were cases of general

suppurative peritonitis. Vogt replied that in the fatal cases the diagnosis made during life was confirmed.

KEYSSER (Lichterfelde) discussed colloidochemical irrigations. He stated that the hydrogen-ion concentration is of the greatest importance since a solution made up according to correct principles in this respect exerts a marked bactericidal effect without injuring the tissues. From this standpoint the ordinary physiological sodium chloride solution is injurious since, by reason of its hydrogen-ion concentration, it favors the growth of bacteria in the tissues. The hydrogen-ion concentration of Ringer's solution is considerably better. Keysser referred to an article he published in the *Klinische Wochenschrift*, 1926, No. 10, in which he stated that the usual disinfectants are made markedly more effective by a suitable hydrogen-ion concentration. The effect of rivanol and of trypaflavine is increased thereby a thousand fold. Keysser has obtained good results with irrigations of such solutions in general suppurative peritonitis. The fundamental solution, the elmocid (that is, a solution found by electro-osmotic methods) is held in readiness and is diluted according to the case and the location in which it is to be used. Of six patients treated with such a solution, all were cured. Two died, but one of these was moribund when first seen and the other died after six days from bronchopneumonia. In the fatal cases the cure of the peritonitis was confirmed by autopsy.

LOEHR (Kiel) discussed perforations of the stomach. He ascribed the failure of operations performed after twelve hours in peritonitis due to perforation of the stomach to the absence of hydrochloric acid which favors the development of pathogenic bacteria. Naumann has rejected this theory, calling attention to the fact that streptococci and staphylococci are present in every stomach, but Loehr emphasizes that these are lactic acid streptococci and a peculiar form of staphylococci which are not hæmolytic. He proved their non-pathogenic character by experiments with pure cultures; on the inoculation of such cultures there was no reaction. In the absence of hydrochloric acid, the colon bacilli come from the small intestine and the hæmolytic streptococci occasionally from the mouth where they are always present. Loehr attempted to clarify this question by experiments on dogs. When the infection was not too severe, he was able to effect a cure of peritonitis by irrigating with a 0.3 per cent solution of hydrochloric acid. The addition of pepsin did not increase the disinfecting power of the gastric juice.

SEELIGER (Freiburg) discussed the treatment of peritonitis. The most common variety of the condition is colon-bacillus peritonitis. In its treatment, it is necessary to destroy the bacillus. The commonly used sodium chloride solution is not able to do this. Ringer's solution, normosal, and glucose solutions are also ineffective. Ether has a bactericidal power but is not satisfactory. However experiments showed that the colon bacillus is killed by twelfth-normal hydrochloric acid solution. Seeliger therefore tried this solution in ten cases of perforation of the appendix, using 3 liters to irrigate the abdominal cavity. All of the cases except one were cured. One patient died of bronchopneumonia. Autopsy revealed a complete cure of the peritonitis and absence of colon bacilli.

ORATOR (Vienna) discussed the insulin-glucose treatment of postoperative shock. He called attention to the fact that the number of deaths following operations is greater on the first day than on subsequent days. This may be explained, in part, by the effect of shock. In shock, intravenous injections of from 100 to 150 c.cm. of a 30 to 50 per cent glucose solution with the addition of from 20 to 30 units of insulin have proved beneficial. The insulin increases the effect. In experiments on animals Orator proved that the sugar blockade of the internal organs is overcome by insulin.

GOETZE (Frankfort) discussed peritoneal infusion in the after-treatment. Irrigation is frequently done at the Frankfort Clinic. The procedure is very methodical. The regions of the hypochondrium are treated first, then the lower structures, and finally the cul-de-sac of Douglas. Attempts at secondary irrigations and at peritoneal infusions in the after-treatment have not proved successful. Moreover, they may break up primary fibrous adhesions and cause a general diffuse peritonitis. Experiments on dogs showed that aseptic as well as non-aseptic peritonitis is aggravated by secondary irrigations.

KUHN (Berlin) discussed hypertonic auto-irrigation. There are two protective forces in the peritoneum—resorption and secretion. Of these, secretion is the most important. It may be produced or increased by the use of hypertonic solutions. The best of these is a concentrated glucose solution. When a 50 to 80 per cent glucose solution is poured into the inflamed peritoneal cavity, a biological and mechanical effect is brought about. The former consists in: (1) an increase in the antagonism of the less harmful sugar-reducing bacterial flora to the various bacteria in the abdominal cavity, which prevents the bacteria that are active in the abdominal cavity from forming toxins, (2) the production of acid products which make the toxins innocuous, and (3) the formation of exudates and transudates with bactericidal powers. The mechanical effects are an auto-irrigation, the isolation of the intestinal loops by the formation of a sugar syrup between them, and a dissolving of fibrin (the solvent action of sugar upon fibrin) which favors drainage. Kuhn recommends the addition of iodine to the glucose solution. He has had good results from the pouring of glucose solution into the abdominal cavity.

NOETZEL (Saarbruecken) agreed with Kirschner that it is impossible to drain the free abdominal cavity. Nevertheless he still uses a drain in the cul-de-sac of Douglas since, in the first twelve hours, it serves to remove any infected irrigation fluid that

may remain in the abdominal cavity. He also still advocates the lowering of the pelvis, but believes that the position need not be as upright as formerly. Up to the present time he still has used camphorated oil, but on the basis of the findings of Seeliger and others, he will hereafter employ solutions of hydrochloric acid instead.

USADEL (Koenigsberg) discussed intestinal activity and the portal circulation. The blood in the general body veins moves through a uniform tubular system that is nowhere especially narrowed, but the blood in the portal system must flow through a second capillary network, that of the liver. Whereas, through suction, the inspiratory reduction of pressure has a favorable effect on the blood circulating in the inferior vena cava, this favorable influence is not present in the portal vein. Here it is the motor activity of the intestine that has a favoring influence on the circulation of the blood.

In order to demonstrate this, Usadel performed experiments on animals in which he measured the amount of blood flowing through the main branch of the portal vein with the Huerthle hydraulic gauge and determined the effect of peristalsis upon the amount of blood flowing through. He found that in normal animals the onset of powerful peristalsis such as may be produced by irrigating large portions of intestine with hot sodium chloride solution results in a threefold increase in the volume of the current. In animals in which a severe peritonitis had been produced and there was hyperæmia of the splanchnic vessels, the extremely retarded blood stream in the portal vein was doubled by the stimulation of peristalsis. Therefore peristalsis of the gut has an undeniable influence upon the amount of blood passing through the portal vein during a certain period of time. This explains why, in free suppurative peritonitis, a decrease in the blood pressure develops only when the intestine loses its automatic activity and meteorism becomes a prominent complication.

Usadel was able to show that, even in normal animals, moderate inflation of the small intestine with air affects the circulation of blood in the intestine in such a degree that the volume flowing through the portal vein per second is reduced to half.

In the treatment of free suppurative peritonitis it is of the greatest importance to prevent the pathological accumulation of blood in the blood vessels of the abdominal cavity. According to Usadel's investigations, the stimulation of intestinal peristalsis is an excellent method for this purpose. In other investigations, Usadel demonstrated the powerful peristalsis-stimulating effect of the sodium chloride infusion, to which Hotz has called attention. This was still further increased by the addition of peristalsis-stimulating and simultaneously vasoconstricting agents such as pituitrin, which has been found very valuable in the Koenigsberg clinic in the treatment of postoperative ileus. Therefore, for the removal of the blood from the portal system in peritonitis, intestinal activity should be stimulated by all possible measures, among which should be included the infusion of sodium chloride solution.

SMIDT (Jena) stated that at the Jena Clinic the decision as to whether irrigation should be done or not is based on two factors, the course of the infection and the nature of the exudate. In general, irrigation and drainage are avoided in peritonitis, especially in cases of serous exudates. In perforations of the stomach and traumatic gastric and intestinal injuries, however, irrigation is done, especially when chyme elements are visible since it is desired to close the abdominal wound primarily. In injuries of the colon irrigation is not done. Smidt called attention to the frequency of peritonitis after appendicitis during epidemics of grippe. Pneumococcus peritonitis is very rare; only two cases have been observed at the Jena Clinic during the last five years.

BREITNER (Vienna) discussed typhoid perforation. He had the opportunity to observe seven cases in a hospital for war prisoners. Operation resulted in a cure in three (43 per cent). The cause of the cure was not the method of operation nor the time at which it was performed (one of the patients who died was operated upon seven hours after the perforation, and one of those who were cured was operated upon nineteen hours after the perforation), but the patient's condition and the stage of the typhoid at the time of the operation. The patients who were cured were in the third or fourth week of the typhoid, and those who died were in the first or second week.

HANS (Barmen) stated that he does not attempt to drain the free abdominal cavity, but drains the focus of infection. Strand drainage is often sufficient to drain a small focus deep in the abdomen. The drains need not be hollow. Hans considers good drainage of the bed of the gall bladder as most necessary. For this, and for drainage of the cul-de-sac of Douglas, he uses suction drainage.

BRUETT (Hamburg) stated that, in general, the same principles as those mentioned by Kirschner are applied in the Hamburg Hospital. In the course of time the mortality has been reduced from 100 to 30 per cent. For irrigation, Bruett prefers hypertonic sodium chloride solution which exerts a good effect on peristalsis. He has avoided the use of hydrochloric acid solutions, fearing that they may have an undesirable chemical effect. The prognosis is influenced by the bacteriological findings. A colon-bacillus peritonitis following appendicitis has a more favorable prognosis than peritonitis caused by anaerobic streptococci. A series of cases of peritonitis due to bacillus aerogenes capsulatus were observed by Bruett in Eppendorf. These were characterized by hæmorrhagic exudate with the odor of a corpse. In peritonitis following the perforation of a gastric ulcer, resections offer a prognosis no more unfavorable than that offered by conservative operations. In twenty-five cases so treated there was only one death.

ROEPKE (Barmen) reported upon the results in 235 cases of suppurative peritonitis. The mortality was only 8 per cent when he operated within the first twelve hours, but rose to 40 per cent when operation was performed after forty-eight hours. Roepke is opposed to the view of Kirschner that the primary establishment of a fistula is to be avoided. He has found that without such relief the abdomen frequently cannot be closed. He considers it indicated especially in the presence of circumscribed paralysis of the intestines. In cases of typhoid perforation, operation should be done as soon as possible. In one case of threatening perforation, Roepke brought the affected coil forward so that the perforation took place externally.

PUST (Jena) discussed biological prophylaxis and treatment. He referred to his demonstration at the previous year's Congress, in which he showed the effect of hot 10 per cent hypertonic sodium chloride solution. Death in peritonitis is due to a sudden overwhelming of the body with toxins. The removal of these outward is therefore of the greatest importance. Hypertonic sodium chloride solution causes a marked transudation. The stronger this is and the more of it that escapes, the better. When the dressings are well saturated the prognosis is good. However, these hot sodium chloride irrigations are of value only in the first stage. In the second stage, when strong adhesions have already formed in the abdominal cavity, they are not effective.

Pust carried out experiments with silicic acid added in finely granular form to the hypertonic sodium chloride solution. These showed that when the silicic acid-sodium chloride solution was introduced into the abdominal cavity simultaneously with infected material, the dogs remained alive whereas the control animals died. When the solution was injected twenty-four hours later than the infectious material, a considerable number of the animals remained alive whereas the control animals died. The silicic acid acts mechanically by plugging the lymphatic stomata and thereby preventing resorption, and has also an adsorptive and lethal effect on bacteria. This is therefore a biological prophylaxis, which should be applied at every laparotomy.

FREY (Koenigsberg) reported upon experiments on animals to determine the effect of the normal intestinal contents upon the development and relief of postoperative, postanæsthetic, and peritonitic intestinal paralysis. On the one hand it is possible that when the intestine is full, intestinal flatulence may result from the decomposition of the intestinal contents and the intestinal paralysis may be so aggravated by the overdistention of the intestinal wall that there is slight chance of relieving it. On the other hand, it is possible that a normally filled intestine is less apt to become paralyzed than an empty intestine and responds better to peristalsis-stimulating remedies after paralysis than an empty intestine, since the normally filled intestine has to its advantage the stimulus exerted by its contents (sensory stimulation of the mucosa and distention stimulus). However, the animal experiments showed that the effect of injuries paralyzing peristalsis and of remedies stimulating peristalsis is independent of the state of fullness of the intestine providing it is within normal limits. The practical conclusion to be drawn from this finding is that emptiness is to be considered the ideal condition of the intestine also from the motor standpoint.

SCHOENBAUER (Vienna) stated that the attempts to influence peritonitis by the introduction of drugs into the abdomen has been abandoned at the von Eiselsberg Clinic. The only exception is pepsin-hydrochloric acid, the use of which has been very satisfactory. In peritonitis due to perforation of the gastro-intestinal tract the mortality was 52 per cent without the use of this remedy, and 26 per cent with its use. In peritonitis after perforation of the appendix the corresponding mortalities were 25 and 8.9 per cent, and in peritonitis due to perforation of the biliary passages, 50 and 0 per cent. The total mortality without the use of pepsin-hydrochloric acid in 164 cases was 34.7 per cent, whereas with its use in 110 cases the mortality was 12.7 per cent. This shows a reduction of the mortality to almost one-third. The postoperative course was also improved by the pepsin-hydrochloric acid. In eight of the fourteen fatal cases no peritonitis was found at autopsy.

NEHRKORN (Elberfall) would add to the groups of gonococcal and pneumococcal peritonitis the peritonitis of infants in which more conservative treatment is indicated. He emphasized the unfavorable prognosis in these cases. He was unable to save the life of one of the children by operation, whereas since he adopted the more conservative plan for children under 2 years of age he has achieved better results. He advocates early operation for other cases, and is in favor of education of the laity with regard to its advantages

RESCHKE (Greifswald) gave the reason for the different stand of Pels Leusden which was mentioned by Kirschner. He stated that because of the unfavorable results obtained in general peritonitis a number of patients who were brought to the clinic in a very poor general condition or apparently moribund were treated conservatively. To the surprise of the clinic staff, all of these patients recovered. Therefore conservative treatment has become the routine treatment in Pels Leusden's cases.

Of 138 patients with general suppurative peritonitis, ninety-nine (71 per cent) died. Of 106 who were operated upon, eighty (75 per cent) died, whereas of thirty-two who were treated conservatively, nineteen (59 per cent) died. From the latter should be deducted seven patients who were brought to the hospital in a moribund condition. When this is done the mortality in the conservatively treated cases is found to have been only 46 per cent. The mortality in cases operated upon on the first day was 33 per cent; that in those operated upon on the third day, 91 per cent; and that in those operated upon after the sixth day, 100 per cent. With

regard to the Kuhn method of pouring hypertonic glucose solution into the abdominal cavity, Reschke claims that the hopes raised by it have not been realized. It was followed by recovery in the early cases, but failed in the late cases.

KOERTE called attention to the fact that the views presented controvert everything that has hitherto been believed by surgeons to be correct. His own experience favors immediate operation after the establishment of the diagnosis.

RESCHKE stated that the Greifswald Clinic is also in favor of early operation in cases recognized early and recommends conservative treatment only in late cases.

ANSINN (Demmin) remarked that the Greifswald procedure with its conservative treatment exerts an unfavorable influence in the vicinity of Greifswald as the patients and physicians are reluctant to resort to operation.

SEYBERTH (Senftenberg) emphasized that there are cases in which the formation of an intestinal fistula is indicated, and that therefore the routine avoidance of this procedure in the treatment of peritonitis is incorrect. He stated also that he observed the percutaneous puncture of the intestines for the first time at the Rehn Clinic. As he was convinced of the good effect of the procedure he has used it since.

KOERTE stated that he fears infection as a result of such punctures.

In conclusion, KIRSCHNER emphasized that he cannot approve of the attitude of Pels Leusden which he regards as a return to the nihilism of thirty years ago. He sees no reason for making the peritonitis of infancy an exception. He expects no improvement in results from Kuhn's glucose solution (lymph lavage), and he is skeptical also regarding the value of the introduction of drugs into the abdominal cavity. In typhoid perforation the most difficult matter is a timely diagnosis. Kirschner believes that the perforation always occurs in the same stage of the condition. For perforations of the stomach he considers conservative measures safe. If the formation of an intestinal fistula is necessary, he would prefer to do it on the following day under local anæsthesia. On the other hand, he has seen good results in several cases from percutaneous puncture of the intestine and therefore favors its use in very severe cases. STETTINER (Z).

Pribram, B. O.: Mesenteric Lymphangeit's (Ueber Lymphangitis mesenterialis). *Arch. f. klin. Chir.*, 1926, cxl, 589.

In numerous cases in which operation is performed for chronic appendicitis, only very slight pathological changes are found in the appendix whereas the lymph channels and glands of the mesenterioleum and mesentery show both the macroscopic and microscopic evidences of an inflammation which has subsided.

On the basis of this observation the author discusses Virchow's suggestion that the infection may progress from within outward, layer by layer, without leaving any evidence of a subsided infection in the intestinal wall.

The hypothesis that in some cases the intestinal wall may show scarcely any inflammatory reaction when bacteria pass through it whereas the lymphatics react with distinct signs of inflammation is based on a varying immunity of different kinds of tissues.

Inflammatory processes in the mesenteric glands, especially those associated with shrinkage, may cause marked symptoms.

In order to prevent the extension of the inflammatory process of acute appendicitis into the lymph channels and glands and the symptoms dependent upon such involvement, Pribram urges early operation.

Another proved cause of mesenteric lymphangeitis is coprostasis. During the war the author noted inflammatory changes in the mesentery in association with megacolon, a condition which is very common in Russia. HOOK (Z).

GASTRO-INTESTINAL TRACT

Koenig, F.: The Treatment of Gastric Ulcer (Zur Therapie des Magengeschwuers). *Muenchen. med. Wchnschr.*, 1926, lxxiii, 51.

Since, in a period of four years, 253 cases of ulcer were operated upon in Koenig's clinic and in a period of seven years only 107 cases were referred for operation by the medical clinic, it is evident that only a small percentage of the cases of ulcer which come to operation are sent from the medical clinic.

Morawitz attributes gastric ulcer chiefly to a continuous spasm of the gastric musculature, one cause of which is the nervous constitution to which Bergmann has called attention. On the basis of research carried on in his clinic by Stahnke, Koenig concludes that the nervous influence causes a gastritis which constitutes the basis of the ulcer.

Whereas Koenig formerly operated only in cases with the classical ulcer syndrome, the indications for operation recognized by him have since been broadened. In some of the cases with the classical syndrome an ulcer was found, but in others only adhesions. One group of patients were relieved of their symptoms by liberation of the adhesions, whereas another group returned with the same symptoms, some with ulcer and others with new adhesions.

In cases without proof of ulcer ih which resection was done at the request of the internist the specimen in every case showed chronic gastritis. On the basis of this finding, Koenig agrees with Morawitz that there is an ulcer sickness without an ulcer. According to the statistics of the medical clinic, only 60 per cent of ulcers can be cured by medical measures, and Koenig's own experience has shown that in a large number of cases the symptoms cannot be relieved without operation.

Koenig formerly favored gastro-enterostomy, but now prefers resection because investigations made over a period of years by Dahl showed that in many cases the good early results of gastro-enterostomy do not persist, and because experience has demonstrated that gastro-enterostomy itself may be harmful. Koenig reports two very instructive cases of peptic ulcer.

In cases of perforated ulcer at the pylorus, resection is best if the operation is done in the first twelve hours. In cases of ulcers at a distance from the pylorus, gastro-enterostomy is unnecessary. Koenig does not deny that an ulcer can be healed by gastro-enterostomy, but he considers this operation only an emergency procedure. The mortality of resection decreases with increasing experience of the surgeon. Koenig uses Reichel's method of resection.

In conclusion, Koenig emphasizes the importance of the proper postoperative care. He states that chronic gastric ulcer is a good example of an ailment in which internal medicine alone is often insufficient and the cooperation of the internist and surgeon is necessary for a cure. STAHNKE (Z).

Lindboe, E.: Experiences with the Resection Method of Pólya in Ulcer of the Stomach and Duodenum (Erfahrungen mit Polyas Resektionmethode bei Ulcus ventriculi und duodeni). *Zentralbl. f. Chir.*, 1926, liii, 1142.

This article is based upon 129 cases in which an operation was performed for ulcer of the stomach or duodenum. In 109 cases the operation was done according to the method of Pólya. In seventy-one the appendix was removed at the same time; it was always found to show a pathological change. In six cases the operation was undertaken because of perforation or hæmorrhage. In ninety of the 101 other cases a retrocolic anastomosis was done and in eleven an antecolic anastomosis. There were four deaths.

In a series of ninety cases treated previously, a subsequent examination showed a cure in 90 per cent and improvement in 4.4 per cent. The mortality was no greater than that of simple gastro-enterostomy. Vicious circle and the later development of carcinoma did not occur. For the prevention of subsequent ulcers it is essential that an extensive resection be performed since, by the removal of the glands secreting hydrochloric acid, the chief causes of new ulcers, hyperacidity and hyperchlorhydria, are decreased. The technique of the operation is simple.

The extent of the resection must be determined from the degree of hyperacidity. Anacidity is better than too great acidity. Gastro-enterostomy should be reserved for cases in which resection cannot be performed. In these cases also the pylorus should be closed off as tightly as possible. Peptic ulcer developed in three of the cases reported, evidently because the resection was not sufficiently extensive. STRAUSS (Z).

Deaver, J. B.: Intestinal Obstruction. *Ann. Surg.*, 1926, lxxxiv, 571.

Deaver attributes the present high mortality in cases of obstruction of the intestines to late recognition of the condition and the administration of purgatives. An early diagnosis requires careful inspection, auscultation, and palpation of the abdomen and a rectal or vaginal examination. The possibility of intestinal obstruction should be borne in mind in every case with acute abdominal symptoms in which the scar of a laparotomy is found. Morphine should not be given before the examination. While purgatives are contra-indicated, attempts to give relief by enemas are justifiable.

Intestinal obstruction occurring three or four days after an operation for acute perforative or suppurative appendicitis is sometimes difficult to differentiate from secondary abscess with circumscribed peritonitis or beginning diffuse peritonitis. The most characteristic symptoms and signs of intestinal obstruction not associated with secondary or residual abscess or diffuse peritonitis are intermittent colicky pain with stormy peristalsis, inability to pass gas, and persistent vomiting. In the presence of such signs, Deaver operates at once.

Paralytic distention of the bowel with regurgitant vomiting with or without hiccough and the absence of characteristic pain simulates intestinal obstruction and late peritonitis so closely that the differentiation is exceedingly difficult.

The condition found is usually leakage of intestinal contents due to causes such as ulcerative perforation of the appendix or of a coil of bowel which at operation for strangulated hernia was thought to be viable enough to recover and was therefore returned to the peritoneal cavity; the separation of a gastro-jejunostomy or entero-enterostomy; the partial or complete opening of the duodenal stump after a subtotal gastrectomy; the escape of duodenal contents after excision and suture of a duodenal or gastric ulcer or the closure of a perforated ulcer without gastro-enterostomy, or the leakage of bile after a cholecystectomy or of urine from accidental incision of the ureter or intraperitoneal rupture of the bladder.

In all of these conditions the symptoms and signs are much the same, including abdominal pain, rigidity, tenderness, slight distention which is more or less general but most marked at the site of the lesion, inability to pass fæces or gas, vomiting, and sometimes hiccough.

Deaver regards it as better to operate and not find a lesion than to wait and then operate and find an obstruction which calls for extensive resection. In every case of acute obstruction there is a peritonitis which in the early hours of the condition is limited, the exudate being merely serous and negative for virulent bacteria. When a patient recently operated upon for an acute abdominal condition and still on a liquid diet develops cramp-like pains and nausea, Deaver orders gastric lavage at once. If the washings have a foul odor and on laboratory

examination are found to contain material from the upper intestinal tract, he operates immediately.

In an occasional case of postoperative acute obstruction of the intestines the condition is attributable to the presence of drains. Therefore the proper disposition and careful charting of drains is essential.

The differentiation between postoperative paralytic ileus and obstruction from kinking is difficult. When Deaver is not sure of the advisability of immediate operation, he prescribes anatomical and physiological rest and if improvement does not occur in a few hours he operates. In his experience, jejunostomy has not been satisfactory in cases of paralytic ileus. In mechanical ileus it is of value if it is done early, but even in this condition a side-track operation is preferable.

In chronic obstruction operation need not be performed immediately unless an acute exacerbation is superimposed on the chronic condition. Chronic obstruction should be treated for ten days by gastric lavage, nourishment by mouth, the administration of normal saline solution with glucose and whiskey by the Murphy drip method, the application of an ice bag to the abdomen, and hypodermic injection of morphine to relieve the pain. During this time a study of the blood chemistry and renal function should be made and circulatory defects corrected or at least treated. Before operation the intestinal tract should be cleared by the administration of mild purgatives, high enemata of sweet oil, and occasionally by cæcostomy.

Chronic obstruction Deaver believes is often of carcinomatous origin. While X-ray examination is important, it is not infallible. Visible peristalsis producing the ladder-rung abdomen is positive proof of the presence of chronic obstruction. Obstruction of the right half of the colon is usually associated with constipation and obstruction of the left half of the colon with diarrhœa. The decision as to whether the operation should be performed in one or two stages can usually be made only after the abdomen is opened. EMIL C. ROBITSHEK, M.D.

Koerte, W.: The Treatment of Acute Mechanical Occlusion of the Intestines (Zur Behandlung des akuten mechanischen Darmverschlusses). *Arch. f. Verdauungskr.*, 1926, xxxvii, 83.

In the author's opinion, paralysis of the intestine should not be called "paralytic ileus" as the term "ileus" comes from the Greek εἰλεά, meaning "to tie up into knots, to twist into a tangle."

It is only in the chronic forms of ileus that there is at first only a stasis of the bowel contents. This leads to injury of the bowel wall secondarily as the result of distention. In such cases an enterostomy may be of benefit.

In acute mechanical occlusion of the intestine there are circulatory disturbances which, if the obstruction is not removed, may be expected with certainty to result in injury of the wall of the bowel. The injurious action of stasis, decomposition of the bowel contents, and resorption of toxic materials occurs only in the further course of the condition. Spastic occlusion of the intestines, which cannot yet be differentiated clinically with certainty from acute mechanical occlusion and is therefore often first recognized at laparotomy, is rare and considerably less dangerous than acute mechanical occlusion of the intestines.

The author divides cases of acute mechanical occlusion with vital indications into those of constriction of the intestine by bands, those of incarceration of the intestine in an intra-abdominal peritoneal sac or defect, those of volvulus, and those of invagination. Gall-stone ileus must also be included in this group since at the point where the gall stone occluded the intestine in the author's cases there were frequently deposits of fibrin on the serosa and in two cases perforation occurred. To this group belong also cases of kinking of the bowel since in these there occurs a mechanical injury to the bowel at the point of kinking. In all of these cases the condition can be benefited, as in cases of incarcerated external hernia, only by operative removal of the constriction.

In only one of the author's cases of invagination, that of a child who came under observation very early, did the condition correct itself spontaneously, the intussusception disappearing with a gurgling sound during examination by palpation. As a rule we cannot count on the loosening up of a constricting band, the untwisting of a volvulus, or the passing of a gall stone without surgical assistance. The results of operation are so often poor because the patients so frequently come to operation late. The earlier the operation is performed the better the results.

Of 236 patients operated upon for acute mechanical obstruction of the intestine, 140 were females. The higher incidence of the condition in the female is explained by the frequency of inflammatory processes of the uterus and adnexa which lead to the formation of bands and adhesions. The total mortality was 43.2 per cent—44.3 per cent in the cases of females and 40.1 per cent in those of males.

At the outset of the condition pain may be absent, but the cessation of intestinal evacuation, increasing vomiting, flaccidity of the abdominal wall, and visible, palpable, and audible peristalsis of the intestine above the point of the occlusion are infallible signs. The diagnosis of ileus can and must be made without the assistance of the X-ray. Wahl's sign, a palpable incarcerated loop which has become distended with gas, is not a certain one except in volvulus of the sigmoid flexure since the point of incarceration becomes covered by the distended loop above it and therefore cannot be palpated. Vomiting occurs later in intestinal occlusion than in peritonitis. Intestinal occlusion that has advanced to the stage of peritonitis often cannot be differentiated from primary peritonitis, but the indications for laparotomy are the same in both conditions. When peritonitis has already developed, the prognosis of operation is unfavorable. When there is intestinal paralysis, the prognosis is very grave.

The character and location of the obstruction can be determined before operation in only a few cases, and even in these only approximately. Invagination is usually recognized before operation because it occurs chiefly in children and is characterized by the appearance of a sausage-shaped tumor and tenesmus with scanty evacuations of blood and mucus. Volvulus of the sigmoid flexure was recognized in several of the author's cases from the palpable loop tensely filled with gas, which extended up into the epigastrium. In cases of gall-stone ileus and adhesions due to inflammatory diseases (appendicitis, parametritis) the history leads at least to a probable diagnosis.

In the author's hospital cases the mortality was 45.5 per cent, whereas in his private cases it was only 20 per cent. The difference was due primarily to the fact that his private patients came to operation earlier. Since in both groups there was a decrease in the mortality in the course of time it is to be assumed that improvement in the operative technique was a factor in the improvement of the results. The patient's age and resistance and the character of the obstruction also play a part in determining the results of operation. Among the patients under 1 year of age the mortality was 60 per cent and in those over 60 years it was 58 per cent.

The factor of chief importance in the outcome of operation is the time at which the operation is performed. In the cases operated upon on the first day the mortality was only 19 per cent, whereas in those operated upon on the second, third, and fourth days it ranged from 38.6 to 33.3 per cent, in those operated upon on the fifth day it was 50 per cent, and in those operated upon on the ninth day it was 100 per cent.

With regard to the character of the obstruction the author states that the most favorable results were obtained in cases of incarceration in peritoneal recesses. In this group, a cure was obtained in 75 per cent. In cases of kinking due to adhesions the incidence of cure was 65.4 per cent, and in those of obstruction from peritoneal bands it was 63.4 per cent. In cases of invagination a cure was obtained in 59.4 per cent; in those of gall-stone occlusion, in 53.4 per cent; and in those of volvulus, in 50 per cent. The poorest results were obtained in nine cases of constriction by bands or volvulus due to Meckel's diverticulum. In this group recovery resulted in only 33.3 per cent.

At operation the author makes an incision from 10 to 12 cm. long in the midline, beginning somewhat above or at the umbilicus and extending it downward toward the symphysis. In acute vital intestinal occlusion an enterostomy without loosening of the obstruction is not sufficient. After the abdomen is opened, the intestinal loops protruding out of the abdominal cavity are enveloped in hot compresses wet with physiological salt solution and are well protected against contact with the iodine skin surface.

A search is then made for the point of obstruction by following the distended loops downward or the collapsed loops upward. Great care is taken to avoid tearing the intestine at the point of the constriction. When possible, the endangered loop is raised out of the abdominal cavity and carefully packed off.

The emptying of the over-filled intestine of its decomposing contents is of the greatest importance. This is accomplished by introducing a thick rubber tube through an incision in an intestinal loop which has been taken out of the abdomen, led through a hole in a large piece of sterile, water-proof cloth, and surrounded by gauze. During the process of emptying, the assistant strips the intestine down toward the opening, beginning with the highest loops. The emptied and sutured intestine is then washed off with physiological salt solution at a temperature of 40 degrees and gently replaced in the abdominal cavity. If the intestine contracts when hot salt solution is sprinkled on it, it is viable.

The intestine emptied of its contents quickly resumes its normal function. Injury to the abdominal contents by this method of relieving the intestine is not to be assumed since in eighty-three cases so treated recovery resulted in 66.3 per cent. Gangrene of the bowel renders the prognosis considerably less favorable.

In twenty-seven cases of intestinal resection for acute occlusion, a cure resulted in only 27 per cent. In some cases, removal of the obstruction was followed by enterostomy and entero-anastomosis as a precautionary measure, but these procedures were not satisfactory and in several instances resulted in fistulæ which required even more radical operations for their closure.

In conclusion the author states that the most important factor in the improvement of the results of treatment is early operation. As regards the operative technique itself he states that improvement in the results is to be hoped for in methodical emptying of the over-filled intestine in an otherwise conservative and faultlessly aseptic procedure.

HINTZE (Z).

Schmieden: Precancerous Diseases of the Intestine, Especially in Polyposis (Praecanceroese Erkrankungen des Darmes, insbesondere bei Polyposis). 50 *Tag. d. deutsch. Ges. f. Chir.*, Berlin, 1926.

Since the best treatment of carcinoma is early operation, it is essential to recognize precancerous conditions. In his studies of polyps, the author discovered a characteristic form of cell which he designates as a precancerous cell. He distinguishes three types of polyps: (1) those which contain normal cells, (2) those which contain precancerous cells, and (3) those in which true cancer nests are present.

According to the findings of the investigations reported, it is incorrect to regard polyps as harmless structures. A specimen should be removed with the proctoscope from every polyp and subjected to careful microscopic examination. Schmieden has known of instances in which the pedicle contained abso-

lutely harmless tissue, whereas further up characteristic precancerous cells were found and in other areas definite cancer nests.

The number of secondary polyps which may contain precancerous cells is much greater than was formerly believed. In some cases the X-ray picture shows them distinctly, but in others only an exploratory laparotomy will reveal their extent and the necessary extent of operation. Roentgen therapy is of little avail in these cases; extensive resection is indicated.

The method of spread of the polyps sheds new light on the question of recurrences and the origin of intestinal carcinoma. Since 60 per cent of polyps undergo malignant degeneration, it seems evident that the cancer has its origin in irritation. The preliminary stages are similar to those assumed by Konjetzny for cancer of the stomach.

In the discussion of this paper, JUENGLING (Tuebingen) reported upon a family followed for several generations, many members of which had either a polyposis or a carcinoma of the rectum.

When Anschuetz inquired whether Schmieden's investigations covered solitary polyps as well as polyposis, Schmieden answered in the affirmative.

ORATOR (Vienna) reported that he had observed malignant degeneration of papillomata also higher up in the intestinal tract. STETTINER (Z).

Harrington, S. W.: Traumatic Retroperitoneal Rupture of the Duodenum; Traumatic Intraperitoneal and Extraperitoneal Rupture of the Duodenum; Strangulated Meckle's Diverticulum in the Right Femoral Canal; Solitary Non-Parasitic Cyst of the Liver. *Surg. Clin. N. Am.*, 1926, vi, 1182.

Harrington reports two cases of traumatic rupture of the duodenum, in one of which the rupture was retroperitoneal and in the other both intraperitoneal and extraperitoneal. In neither case was there any evident injury of the abominal wall, but in the first one a small rupture of the fascia, muscle, and peritoneum was discovered toward the close of the operation. Both patients were in a state of shock and neither recovered.

In the first case a large crepitant hæmorrhagic mass was found in the root of the mesentery and over the second portion of the duodenum at the site of a linear opening about 5 cm. in length, through which food and duodenal contents were draining into the retroperitoneal tissues. Convalescence was fairly favorable until a duodenal fistula developed.

In the second case there was no pain so long as the patient was not disturbed, but palpation revealed marked tenderness in the right upper quadrant. This patient was seen almost immediately after the accident, but the leucocytes numbered 22,000 in comparison with nearly 28,000 in the first case. Thirst was the chief complaint. Bile-stained blood, mucus, and food particles were found in the abdominal cavity, but exploration revealed a small perforation on the anterior wall of the second part of the duodenum and a considerably larger perforation in the posterior wall. There was also considerable traumatization of the intervening tissue The liver was ruptured in two places and the spleen in several. For six hours after the operation the condition was fairly good, but the pulse and temperature rose and death occurred after twelve hours.

The author attributes injury of the duodenum, which occurs in 10 per cent of the reported cases of traumatic rupture of the intestinal tract, to the fixed position of this portion of the intestine in front of the spine. The early leucocytosis is probably attributable to both the intra-abdominal hæmorrhage and the contamination.

Moffitt, H. C.: The Medical Aspects of Duodenal Ulcer. *Canadian M. Ass. J.*, 1926, xvi, 1044.

Starr, F. N. G.: The Surgical Treatment of Duodenal Ulcer. *Canadian M. Ass. J.*, 1926, xvi, 1051.

Dickson, W. H.: The Radiological Aspect of Duodenal Ulcer. *Canadian M. Ass. J.*, 1926, xvi, 1053.

MOFFITT states that today the surgeon and internist see the main problems of duodenal ulcer from a common viewpoint. Certain factors and phases in the etiology, physiology, and pathology of the lesion remain obscure, but definite knowledge has been gained as to the course, symptoms, and treatment.

Ulcers may be produced experimentally by mechanical, chemical, or thermic irritation, by resection of the vagi or splanchnics or of the adrenals or parathyroids, and by the local or intravenous injection of chemicals, bacteria, or bacterial or metabolic toxins.

Many ulcers in man heal promptly without symptoms. The formation of the chronic "clinical" ulcer requires definite injury to the cells of the mucosa and the action of certain forces inhibiting repair of the damaged tissues. Ischæmia due to excessive and persistent local muscle spasm may play a part.

The profound influence on the digestive mucous membrane of toxins or bacteria reaching it by way of the blood stream has been proved conclusively by experimental and clinical evidence in recent years.

Rosenow says that streptococci, irrespective of their source, exhibit, when of a certain grade of virulence, an affinity for the gastric mucous membrane and when injected intravenously may cause an ulcer of the stomach or duodenum.

The healing of an ulcer is inhibited by many forces, among which are fatigue, exposure to cold, long-continued nervous strain and worry, chronic infection, and bad habits which lower the general resistance. McCarrison noted the development of both gastric and duodenal ulcers in monkeys and guinea pigs fed on diets deficient in vitamines. Although ulcer may be formed and may persist with absence of free acid or even with achylia, it has been recognized since the time of Celsus that peptic digestion plays a large part in keeping active the so-called chronic or clinical ulcer.

Duodenal ulcer is a common lesion, at least six or eight times as common as gastric ulcer. Multiple ulcers are recognized with increasing frequency. The extension of the inflammation without perforation may result in abscess or in adhesions. Abscess is rare, but adhesions are exceedingly common. Congenital abnormalities of the duodenum may be found associated with ulcer.

Among the most important complications are stenosis of the pylorus from spasm, inflammatory œdema, and the formation of firm scar tissue. The most striking clinical feature of duodenal ulcer is periodicity. A carefully taken history will reveal this in from 80 to 90 per cent of cases. Early symptoms may be unnoticed or obscure or so abrupt and severe as to render the picture confusing, but as time goes on cycles of activity are marked by the most characteristic events and succeeded by intervals of latency in which the patient may be able to eat any kind of food without any disturbances.

The lesion is uncommon in children and young adults. This may be explained by the greater freedom from infection, nervous and physical strain, and bad habits and the greater activity of the defense mechanism such as the reticulo-endothelial system in the young as compared with the old.

Perforation, when acute, is practically always the rupture of an ulcer on the anterior wall. There seem at times to be epidemics of acute perforating ulcers. Vomiting may be an occasional sign, but when it is persistent should suggest another diagnosis or complications, especially obstruction. X-ray examinations are invaluable and should be made to supplement the clinical investigation. Negative evidence should lead to a careful review of the clinical diagnosis. Excessive secretion of high acidity, food remnants from the night before, and sarcines or yeasts in large numbers in the sediment indicate obstruction of low or high degree.

In the differential diagnosis the periodicity of the pain, its diurnal variations, and its modification by various factors are of great significance. Marked changes in the familiar sequence of symptoms may indicate complications such as obstruction and adhesions or of the development of some other abdominal condition. Frequently the onset of symptoms occurs soon after an abdominal operation. As a rule the patient with ulcer has had insufficient medical treatment.

On the basis of our present knowledge, the treatment aims at the removal of causes of nerve irritation and muscle spasm, the control of infection as far as possible, and the neutralization of gastric juice corrosion. In preventive treatment, regulation of the patient's habits, care of the nervous system, frequent meals, the judicious use of drugs such as alkalies, bromides, and atropine, are indicated.

It is nearly unanimously agreed that in the early stages of the condition medical treatment should be given a trial. The initial treatment should be thorough. Ambulatory treatment may alleviate the symptoms but is less apt to effect a cure.

The experience of surgeons indicates that partial perforation occurs in about 20 per cent of duodenal ulcers. The pancreas may be deeply eroded and chronic pancreatitis may result. The author knows of two cases in which diabetes developed. Free perforation into the peritoneal cavity is prevented in most cases by the omentum or adjacent viscera.

The patient may complain of pain of a gnawing, burning, or boring character which frequently becomes sharp and cramp-like. This pain may radiate to the right or left or to the back, but as a rule is most severe in the upper mid-abdomen. Once established, it exhibits remarkable uniformity, regularity, and what Moynihan has termed "punctuality."

The importance of a careful study of the sequence of events in a twenty-four-hour period during the active stage has been particularly emphasized by Sippy and Eusterman. The condition is characterized by absence of symptoms when the stomach is empty, the occurrence of pain from one to three hours after the ingestion of food, the relief of the pain by food, its recurrence in greater severity from two to three hours after the midday meal, and its relief when the stomach becomes empty or when food is taken. The cessation of the pain after the administration of alkalies, vomiting, and gastric lavage, its modification by rest and compresses, its aggravation by certain foods, exposure to cold, and nervous and physical strain offer important diagnostic aid. Hæmorrhage is much less frequent in private patients than in hospital patients, probably because in the former the disease is recognized earlier and the complications are therefore fewer.

The most rational scheme of medical treatment yet evolved for duodenal ulcer is that of Sippy. Its principles are sound and its results encouraging. We have learned the dangers of the excessive administration of alkalies, especially in cases of retention or coincident renal or hepatic disease.

Indications and contra-indications for operation should be studied carefully by both the internist and the surgeon since, as Eusterman has noted, poor results from operative treatment are due more often to a careless selection of cases than to poor surgery. It is the duty of the internist to prepare the patient properly for operation. Lavage, transfusion, control of alkali dosage, the administration of glucose by rectum or intravenously, and hypodermoclysis of 1 per cent salt solution are measures which have contributed enormously to favorable operative results.

Surgeons have learned to appreciate the value of postoperative treatment. It takes weeks or months to heal a chronic ulcer, even after gastro-enterostomy.

STARR states that duodenal ulcers following severe burns are becoming very rare because of the improved treatment of burns by the immediate application of a 5 per cent tannic acid solution, direct blood transfusion to combat shock, and exsanguination transfusion.

Perforating duodenal ulcers must be recognized and operated upon early. Cautery or knife excision with closure of the opening is sufficient. In cases of penetrating ulcers with marked inflammatory reaction it is necessary to do a posterior gastro-enterostomy in addition to the cauterization of the ulcer in order to keep the patient from starving to death. The gall bladder and appendix should be examined for infection. Duodenal ulcers associated with active hæmorrhage should not be operated upon. In such cases 5 c.cm. of calcium chloride and, if necessary, a transfusion may be given.

In cases of ulcer of the anterior surface of the duodenum, Starr cuts the pyloric ring and performs some type of pyloroplasty. In cases of large callous ulcers producing complete obstruction he does a partial gastrectomy. The duodenum is closed and an anterior Pólya or a posterior gastro-enterostomy is done.

After the operation the patient is watched for the development of alkalosis or acidosis and is treated accordingly. When the patient is discharged from the hospital he is given instructions as to diet and general hygiene.

DICKSON calls attention to the fact that gall-bladder disease, carcinoma of the pylorus, appendicitis, renal calculus, tabes, duodenal ileus, and abnormalities of the ligament of Treitz may give rise to symptoms identical with those of duodenal ulcer. In such cases a careful X-ray examination is of the greatest importance in the diagnosis.

The competent roentgenologist is able to make a correct diagnosis of duodenal ulcer in from 93 to 95 per cent of the cases. About 90 per cent of duodenal ulcers occur in the first portion of the duodenum, the caput duodenalis, a fact of great advantage to the roentgenologist as this portion of the duodenum fills better and remains filled longer than the other portions.

There are two methods of examination. The first depends almost entirely on the use of the fluoroscope; roentgenograms are made only for record. This method is used throughout Europe and in some American clinics. The second method consists in the making of serial roentgenograms as suggested by Cole. In this procedure no screen is used. The number of roentgenograms made ranges from thirty-five to fifty. Dickson has combined the two methods. After screening the patient in the upright and horizontal positions he makes from twelve to fifteen plates.

Deformity of the caput of the duodenum is the direct evidence sought. This is caused by the crater of the active ulcer, by fibrous or scar tissue in a chronic or recurring ulcer, and, in some cases, by spasm. The most common deformity caused by an ulcer is the formation of the clover leaf cap. A crescentic or bud-like projection is often observed protruding from the lesser curvature side of the caput. This is due to the crater of the active ulcer in which the mucosa and part of the muscularis are destroyed and the defect is filled with the barium.

Chronic duodenal ulcer may produce so much scar tissue that the caput is virtually obliterated and cannot be visualized. In such cases we must depend to some extent on indirect evidence such as hyperperistalsis and retention to establish the diagnosis. In cases of chronic perforating ulcers due to localized peritonitis, X-ray examination will reveal an accessory pocket. The outline of the pocket is usually irregular and the fistulous tract leading to it may or may not be visible. A duodenal diverticulum may be confused with an accessory pocket. The latter, however, is usually smooth and round and contains no gas or secretion.

Deformity of the caput is often caused by adhesions of gall-bladder origin. In such cases the caput will be found somewhat angulated upon the pyloric end of the stomach by the drag of the adhesions. The dilated gall bladder itself causes deformity of the caput, but can be differentiated by the fact that it presents a smooth round pressure defect in the latter.

The indirect signs of duodenal ulcer are hypertonicity with increased peristalsis and decreased emptying time in the non-obstructive or early obstructive cases, and decreased peristalsis with increased emptying time in cases of long-standing obstruction. Reflex duodenal spasm may be excluded by the use of antispasmodics or by keeping the patient on a milk diet for several days preceding the examination. MERLE R. HOON, M.D.

Mayo, C. H., and Powell, L. D.: The Colon as a Urinary Receptacle. *Surg. Clin. N. Am.*, 1926, vi, 1131.

Mayo and Powell discuss transplantation of the ureter into the sigmoid and report three cases in which the operation was performed by Coffey's method. They believe that the operation might be widely employed in cases of severe injury and loss of substance or control of the sphincters, as well as in cases of cancerous growths of the bladder.

Altogether they have seen about 100 cases of exstrophy of the bladder. In six, cancer of the bladder had developed. The operation of transplantation should not be performed before the age of 5 years.

The first case reported was one of epispadias in a boy aged 13 years. The left ureter was transplanted thirteen days after the right ureter. Rectal control of the urine was excellent.

The second patient was a woman 27 years of age. Exstrophy of the bladder was complicated by severe hydronephrosis of the left kidney with obstruction of the left ureter. The right ureter was transplanted into the sigmoid with a good result. More than a year later the patient complained of pain in the left side of the abdomen. As the left kidney was found to be functionless, it was excised with its ureter. In this case the symphysis pubis was missing and both uterus and vagina were double.

In the third case, that of a woman aged 39 years, exstrophy of the bladder was associated with right

hydronephrosis and carcinoma of the bladder. The right kidney was removed and the left ureter transplanted into the sigmoid a month later. Urine was immediately excreted through the rectal tube. Ten days later the carcinoma was removed. The subsequent course has been satisfactory.

Kelling: The Question of Unsuccessful Appendectomies (Zur Frage der vergeblichen Appendektomien). 50 *Tag. d. deutsch. Ges. f. Chir.*, Berlin, 1926.

In a large percentage of cases the pain for which an appendectomy was done persists after the operation. This occurs most frequently when the pathological changes are slight. It is evident, therefore, that in such cases there must have been an error in the diagnosis.

Kelling calls attention to the fact that in young persons the cæcum lies much higher than in older persons and under the weight of its contents gradually sinks down. When there is atony of the abdominal wall or of the pelvic floor and when the patient is emaciated the sinking of the cæcum causes a dragging pain which is very similar to that of chronic appendicitis. This occurs most frequently in asthenic persons and persons whose occupation demands considerable walking or standing. It is more common in girls than in boys. Atony and flatulency of the cæcum often results.

As treatment, the author recommends rest in the recumbent position, bandaging, and the administration of light narcotics to decrease nervous irritability. In some cases the pain ceases spontaneously after a time because the static equilibrium in the abdominal cavity becomes adjusted.

In the discussion of this report, KAUSCH (Berlin) recommended the removal of the appendix at every operation in its vicinity. The patient should be informed that this was done. Kausch believes the appendix should be removed also whenever the patient expects to take a long journey during which he will not be near a surgeon. He makes a small incision in order that there will be few adhesions. He called attention also to the picture of ileocæcal pyæmia which he has seen frequently of late. This is due to a thrombophlebitis which should be treated surgically by the method of Martens.

STETTINER (Z).

Carlson, G. W., and Marshall, V. F.: Intestinal Obstruction Following Appendectomy. *Ann. Surg.*, 1926, lxxxiv, 583.

The authors review twenty-one cases of obstruction of the intestines following appendectomy. Fifteen of the patients were males. In all, the obstruction was due to adhesions. Perforation of the appendix had occurred in eleven cases. In eight, there had been abscess formation, and in six, the appendix had been gangrenous.

The clinical picture caused by the obstruction was not always constant or characteristic. The time of onset of the obstruction varied, but in the majority of the cases the symptoms made their appearance during the first two weeks after the operation. The early manifestations were peristaltic pain, a slight watery regurgitation and vomiting, distressed facies, and constipation.

There were six deaths, a mortality of 28.5 per cent. A favorable prognosis is entirely dependent upon early diagnosis followed by prompt surgical interference. EMIL C. ROBITSHEK, M.D.

Lundy, J. S., and McCuskey, C. F.: Sacral Anæsthesia in Operations on the Rectum. *Atlantic M. J.*, 1926, xxx, 14.

Sacral anæsthesia is the state of analgesia which may usually be obtained by one of three procedures: caudal, presacral, or transsacral block. In a series of 617 cases a combination of caudal and transsacral block was used.

The cases were grouped, according to the diagnosis, as good, fair, poor, or necessarily combined. In the cases classified as "poor," no other form of anæsthesia was used to complete the operation. In the "necessarily combined" group, light general anæsthesia was resorted to in order to overcome pain and nervousness or because of an unexpected increase in the scope of the operation. When regional and general anæsthesia was planned from the outset, the group was designated as "intentionally combined." These cases were excluded from the tables of percentages of successful regional anæsthesia.

Marked untoward reactions were avoided by making the injections slowly. Untoward reactions to the anæsthetic were graded as slight when there was a momentary variation in the pulse rate, a tremor, pronounced palpitation, and slight nausea. These symptoms occurred alone or in combination. When they became more pronounced a marked reaction was recorded.

Satisfactory anæsthesia without untoward results is ascribed to careful attention to minor details of technique.

The technique which is carried out under aseptic conditions is described in detail. A 1 per cent procain-epinephrin solution was used as a routine. From 20 to 45 c.cm. of this solution was injected into the caudal canal, the amounts being decided by such factors as the scope of the operation and the patient's tolerance and body weight. The number of sacral foramina injected was determined by the operative procedure intended. The total amount of solution used in the foramina ranged from 20 to 38 c.cm.

It is important that the caudal needle be within the caudal canal. This is demonstrated by: (1) the aspiration of blood or spinal fluid; (2) the free flow of fluid from the syringe to the canal; (3) a change in the respiration, usually an increase in the depth of respiration as the solution is being injected; (4) tingling or hyperæsthesia referred to the thighs or legs and a sensation of pressure in the sacrum; (5) often a slight change in the pulse rate; and (6)

early anæsthesia of the skin over the coccyx followed by anæsthesia of the perineum and absence of the anal reflex.

In three or four cases in which the caudal canal was not found, transsacral block alone was employed.

In 382 cases of hæmorrhoidectomy, sacral anæsthesia was satisfactory in 95 per cent, fair in 3.4 per cent, poor in 1.3 per cent, and intentionally combined in 0.2 per cent.

In the miscellaneous group of 255 cases, which included minor rectal surgical diseases, the results were satisfactory in 96.8 per cent, fair in 1.1 per cent, poor in 1.9 per cent, and intentionally combined in 0.7 per cent.

In eighty cases of posterior resection of the rectum the lesion was high in sixty-four cases and low in sixteen. Of the cases of high lesions, the anæsthesia was satisfactory in 74 per cent, fair in 7.4 per cent, necessarily combined in 18.5 per cent, and intentionally combined in 15.6 per cent. Of those of low lesions, a satisfactory anæsthesia was obtained in 85.7 per cent. In 14.2 per cent the result was fair, and in 12.5 per cent the anæsthesia was necessarily combined.

In three patients of this series spinal anæsthesia was produced as the result of puncture of the tip of the dural sac with the caudal needle.

Frequently a patient who is a poor subject for local anæsthesia may be converted into a good risk by the judicious use of morphine in amounts sufficient to quiet him without jeopardizing his safety. In the cases reviewed a preliminary hypnotic of ⅙ gr. of morphine sulphate and 1/150 gr. of atropine sulphate, or 1 gr. of codeine was given.

Failure to anæsthetize is due usually to failure to inject the procain-epinephrin solution into the caudal canal or to the starting of the operation before anæsthesia has become established.

LIVER, GALL BLADDER, PANCREAS, AND SPLEEN

Bollman, J. L., Sheard, C., and Mann, F. C.: The Surgical Significance of Experimental Studies Concerning the Sites of Formation of Bile Pigment. *Surg. Clin. N. Am.*, 1926, vi, 1257.

The article is begun with a brief presentation of the three classes of jaundice (obstructive, hepatic, and hæmolytic), the van den Bergh test for bilirubin, and the difference in the character of the reaction (immediate or delayed) depending upon whether or not the jaundice is due to obstruction.

The authors deal especially with spectrophotometric methods, their use in the determination of bilirubin and their application to studies on the sites of bilirubin formation, the formation of bilirubin from hæmoglobin, and the relative importance of the liver, spleen, and bone marrow in the formation of bile pigment.

The spectrophotometric method is about fifty times as sensitive as the van den Bergh reaction. By its use, differences of 0.002 mgm. for each 100 c.cm. can be quite accurately determined. The blood from hepatectomized dogs contains a small amount of yellow pigment which spectrophotometric determinations show has the same characteristic absorption as that of bile from the gall bladder of a normal animal. Since this identity exists, it is evident that the bilirubin formed outside of the liver is identical with the bilirubin of the bile.

It was found that the blood returning from the spleen in the splenic vein always contains a measurably greater amount of bilirubin than the blood of the splenic artery, a fact indicating that the spleen must be considered a site of bilirubin formation. Samples of blood from the femoral arteries and veins and other venous blood obtained directly from the muscle and skin of the extremities demonstrated that bilirubin is added to the blood in the bone marrow, and that little, if any, bilirubin is formed in other tissues of the extremities. After splenectomy, ligation of the common bile duct, and removal of the gall bladder, the blood leaving the liver and free from dilution with other blood, showed that bile pigment is formed in the liver.

The injection of laked blood into the splenic artery produced a definite increase in the bilirubin content of the blood coming from the spleen. This was true also of injection into an artery leading to an isolated bone. After ligation of the common bile duct and extirpation of the gall bladder, the injection of laked blood produced a marked increase in the rate of accumulation of bilirubin in the blood and the appearance of a substance which was probably hæmatin. It seems quite evident, therefore, that hæmoglobin is converted into bilirubin in the usual sites of bilirubin formation.

A series of experiments on normal dogs to determine the relative importance of the spleen, liver, and bone marrow in the formation of bile pigment showed that, while bilirubin is made in both the spleen and the liver, the amount is insignificant as compared with that made in the bone marrow. More bilirubin appears to be formed in the spleen than in the liver.

In conclusion, the authors state that bile pigment is apparently a waste product formed during the destruction of red blood cells. The retention of bile pigment in the blood and tissues in jaundice is similar to the retention of urea in uræmia, and the factors which produce retention of urea in urinary disturbances seem to have their analogue in bile-pigment retention in biliary diseases.

Wildegans: Exerimental and Clinical Investigations in Cholæmia (Experimentelle und klinische Untersuchungen bei Cholaemie). 50 *Tag. d. deutsch. Ges. f. Chir.*, Berlin, 1926.

In experiments on dogs the author attempted to answer the following questions:

1. What is the condition of the clotting elements of the blood (thrombin and fibrinogen) in occlusion of the common duct and following the intravanous injection of bile and the direct conduction of bile

into the blood by transplantation of the common duct into the inferior vena cava?

2. What relationship is there between the clotting elements and cholesterinæmia, bilirubinæmia, bile acids, and liver function?

The thrombin and fibrinogen were determined quantitatively. After obstruction of the common duct there was a marked and very rapid decrease in the thrombin. The activators disappeared from the blood in from three to four weeks, but the fibrinogen content showed little change. The cholesterin increased to four or five times the normal in the first three or four weeks, but then decreased. The bilirubin content of the blood did not increase progressively but receded at the end of the fourth week, whereas, when the obstruction continued, the mechanically and chemically changed liver cells produced progressively less bile and pigment.

It was impossible to determine the quantities of bile acids that circulated in the blood of the cholæmic animals, but it is certain that the smaller the quantity of bile acids excreted into the blood the longer the icterus persisted.

If the bile and bile constituents circulating in the blood have a direct influence upon the blood or blood vessels, this effect would be expected when the bile constituents accumulate in the blood. Such, however, is not the case. Disturbances in the clotting system are most apt to occur when the function of the liver is reduced. This is in agreement with the fact that the intravenous injection of bile and the implantation of the common duct into the inferior vena cava caused no demonstrable disturbance in the clotting process. Liver insufficiency in obstruction of the common duct is manifested by a distinct disturbance of the function of urea formation.

On the basis of his clinical investigations the author recommends the quantitative determination of thrombin in cases of icterus in which operation is contemplated. The procedure, which is simple, furnishes valuable information with regard to the condition of the clotting processes, and, when the activators are deficient, makes it possible to increase the thrombin elements in the blood by the introduction of normal blood. WILDEGANS (Z).

Pancoast, H. K.: The Roentgenological Diagnosis of Liver Abscess with or without Subdiaphragmatic Abscess. *Am. J. Roentgenol.*, 1926, xvi, 303.

Having had the opportunity to examine an unusual number of cases of hepatic abscess during the past three years, and having failed to render an exact diagnosis in many of them, the author made a study of all new cases and of cases of other conditions which were difficult to differentiate. As a result of this study, he believes that he has evolved a process of reasoning and interpretation which makes the diagnosis of liver abscess possible in most instances and renders the roentgen examination of value to the surgeon.

In the type of cases discussed, the important avenue of infection of the liver is the portal circulation or the biliary ducts. A correct history of the case is of paramount importance since upon it must be based the diagnostic procedure and the interpretation of the findings. Roentgenoscopy is absolutely essential. Single flat bedside films have little diagnostic value. Pneumoperitoneum may be of value in some instances, but is entirely unnecessary and would be impracticable in serial study, which is so often required, and might be very dangerous.

Numerous cases are cited in detail with roentgenograms illustrating the findings.

ADOLPH HARTUNG, M.D.

McIndoe, A. H.: Intrahepatic Lithiasis Associated with Multiple Internal Biliary Fistulæ. *Surg. Clin. N. Am.*, 1926, vi, 1233.

McIndoe reports a case of intrahepatic cholelithiasis associated with an internal biliary fistula between the gall bladder and colon and the duodenum and cystic duct. On analysis, the stones proved to be bilirubin-cholesterin stones containing no calcium. The terminal condition was acute suppurative cholangeitis with multiple abscesses in the right lobe of the liver. The patient had never suffered pain although the ducts were crammed with stones, and until the onset of the acute cholangeitis, had been only slightly jaundiced.

Duchinova, S. I.: Temporary Clamping Off of the Hepatoduodenal Ligament for Bloodless Operations on the Liver (Ueber temporaere Abklemmung des Ligamentum hepato-duodenale fuer blutlose Operationen an der Leber). *Vestnik chir. i pogranič'nych oblast'j*, 1925, v, 34.

From experiments on dogs with regard to the effect of clamping of the hepatoduodenal ligament, the author draws the following conclusions:

1. Dogs cannot survive one hour's stagnation of the blood in the portal system.

2. After clamping for five minutes, the liver becomes quite exsanguinated. The method is therefore a very good hæmostatic procedure.

3. After the clamping, the blood pressure sinks, especially in the first five or ten minutes. The degree of the decrease and the return to normal vary greatly in different animals.

4. If the clamping period exceeds thirty-five minutes, the vasomotor apparatus is severely injured.

5. The chief cause of the fall in the blood pressure with functional disturbances of the central and peripheral vasomotor apparatus seems to be the clamping of the portal vein. The isolated clamping of this vein is followed by a less marked fall in the blood pressure than the clamping of the entire hepatoduodenal ligament, but is not sufficient for a bloodless operation.

6. Shutting off of the arterial current lowers the blood pressure by half. A decrease is noted also when the common duct is clamped.

7. Subcutaneous injections of sodium chloride solution do not prevent the fall in the blood pressure. Clamping of the superior and inferior mesenteric arteries is too severe a measure for a weakened patient and is followed by a marked fall in the blood pressure. The fall in the blood pressure can be overcome only by compression of the aorta below the diaphragm for from thirty to forty minutes.

8. Clamping of the hepatoduodenal ligament for half an hour does not cause any special functional disturbances in the circulatory organs, kidneys, pancreas, liver, or intestines.

9. In the pathogenesis of the fall in the blood pressure which follows clamping of the hepatoduodenal ligament the mechanical stoppage of the circulation plays an important part. Intoxication is also a factor, but in the experiments reported no reflex effects were noted.

10. Toxæmia is recognized by the demonstration of vasodilating substances.

11. Clamping of the hepatoduodenal ligament is not a harmless procedure but may be done under precautions in cases of profuse liver hæmorrhage.

ALIPOV (Z).

Higgins, G. M., and Mann, F. C.: Consideration of the Gall Bladder with Reference to the Process of Emptying. *Surg. Clin. N. Am.*, 1926, vi, 1241.

Recent studies on the physiology of the gall bladder have, in large part, dealt with the manner in which the vesicle empties. Factors that have appeared to students of the problem to be instrumental in the process are the secretory pressure of the liver, the intra-abdominal pressure, the respiratory squeeze, the tonicity of the common duct sphincter, and the contractility of the intrinsic muscularis of the gall-bladder tunic.

Following a diet of egg yolk and cream, the gall bladders of animals invariably empty through the cystic duct within an interval of from three to five hours. Studies on certain fish, amphibia, birds, and mammals have proved this to be true. Experiments on guinea pigs are reported wherein the gall bladder was isolated from the peritoneal cavity and watched for an interval following the intraduodenal administration of the test diet. Under these conditions the gall bladder partially empties by the contraction of independent muscle areas, gradually changing its shape and reducing its content of bile through the common duct.

Local anæsthesia permits a study of the normal gall bladder of the dog undisturbed by the effects of ether. By ligating all hepatic ducts and intubating the common duct, it is possible to study the intrinsic movements of the gall bladder following a fat diet and to record these by means of a manometer and drum.

The authors concluded that the gall bladder empties by the contraction of its own intrinsic musculature; that the secretory pressure of the liver is of no great importance in the emptying of the vesicle; that the intra-abdominal pressure is not an important factor in the emptying of the vesicle, and that the sphincter of the common duct is not an inhibitor to the flow of gall-bladder bile.

Stewart, W. H., and Ryan, E. J.: Further Advancements in the Technique and Interpretation of Cholecystography by the Oral Method. *N. York State J. M.*, 1926, xxvi, 819.

Because of many disastrous results from the intravenous use of tetrabromphthalein and tetra-iodophenolphthalein, the authors set about to perfect the oral method. At present they have adopted the following technique:

The intestinal tract is thoroughly cleansed by the administration of a cathartic and an enema. At 6:30 p.m. a light meal is given. Beginning at 9:30 p.m., two 5-gr. capsules of tetra-iodophenolphthalein are given every fifteen minutes with a small glass of water until 40 gr. have been taken. The dye is given in plain gelatine capsules which are dipped into a solution of keratine to seal them air tight and covered with a coating to prevent them from being broken up in the stomach. These capsules are made up fresh for each patient.

The patient reports without breakfast at 9:30 a.m., twelve hours after the administration of the dye. Four hours later, at 1:30 p.m., the examination is repeated. A regular luncheon is then given and another examination is made one hour later. The following morning the final observation is made.

The authors state that this technique can be relied upon absolutely, and that attention must be centered on the interpretation. Non-opaque stones are visualized as circular negative shadows. Mottling of the shadow indicates small stones, but care must be taken to distinguish these from gas in the duodenum. Not all stones are visualized since calcium stones which show with the ordinary method of examination are overshadowed by the opaque bile. Stones may not show when they are overshadowed by the opaque bile in addition to the shadow cast by the thickened gall-bladder wall. Deformity of the gall bladder can be demonstrated. Changes in the function of the gall bladder are indicated by the absence, persistence, faintness, or late appearance of the shadow. The lack of sufficient opaque bile to cast a shadow may be due to impairment of the function of the liver. Faintness of the shadow is attributed to insufficient concentrating power of the gall bladder.

JOHN A. WOLFER, M.D.

Graham, E. A., Cole, W. H., Copher, G. H., and Kodama, S.: Some New Phases of the Physiology of the Biliary Tract. *Ann. Surg.*, 1926, lxxxiv, 343.

Richardson, E. P.: Surgical Aspects of Certain Phases of Liver Function. *Ann. Surg.*, 1926, lxxxiv, 352.

McGuire, E. R.: Problems in Gall-Bladder Surgery. *Ann. Surg.*, 1926, lxxxiv, 366.

Cave, H. W.: Dangers Incident to Cholecystectomy. *Ann. Surg.*, 1926, lxxxiv, 371.

Deaver, J. B., and Burden, V. G.: Surgical Management of the Complications of Cholecystitis. *Ann. Surg.*, 1926, lxxxiv, 379.

Bruce, H. A.: Association of Cholecystitis with Duodenal Ulcer. *Ann. Surg.*, 1926, lxxxiv, 387.
Douglas, J.: Strictures and Operative Injuries of the Bile Ducts. *Ann. Surg.*, 1926, lxxxiv, 392.
Crile, G. W.: The Operative Management of Common Duct Stones. *Ann. Surg.*, 1926, lxxxiv, 411.

GRAHAM, COLE, COPHER, and KODAMA state that the gall bladder has two known functions, one of which is concerned with the regulation of pressure within the biliary tract and the other with the concentration of bile by the absorption of water. As the blood from the gall bladder empties into the portal vein, it is possible that, in addition, the gall bladder forms some product required by the liver.

With regard to the manner in which the gall bladder empties itself, the authors state that if contractures occur they are of little importance. They believe that the bile is siphoned from the cystic duct by the passage of bile down the hepatic or common duct.

The existence of a true sphincter of Oddi is questioned, but a sphincter action produced by the tonus of the duodenum has been demonstrated. The bile is eventually drawn into the bowel by the milking action of the duodenum. Oleic acid or a meal of fatty food is more potent than magnesium sulphate in causing a discharge of bile from the gall bladder. In experiments on animals in which a rubber bag was substituted for the gall bladder, it was found that the bag responded in the same manner as the organ with the exception of the absorptive processes.

For cholecystography, phenoltetra-iodophthalein possesses many advantages over tetra-iodophenolphthalein.

RICHARDSON cites the work of Mann and McGath which has demonstrated a decrease in the blood sugar and urea and an increase in the blood uric acid following the removal of the liver. In hepatic disease the blood chemistry seems to be of more value in revealing the condition of the kidneys or secondary changes in metabolism than it is in revealing the condition of the liver itself.

The bile pigment is probably formed in the recticulo-endothelial system from the destruction of hæmoglobin. It is then excreted by the epithelial cells of the liver. The depth of jaundice depends upon the rate of red cell destruction, the condition of the liver, and the ability of the kidneys to excrete the pigment. The van den Bergh reaction and the icterus index are of value in revealing the degree of latent and clinical jaundice.

According to McNee, jaundice may be caused by excessive hæmolysis, toxic or infectious action on the liver epithelium, the administration of certain drugs, or obstruction of the bile passages.

The clinical picture is considered of more value as evidence for or against operation than the van den Bergh reaction. The chief value of the phenoltetra-iodophthalein test lies in the fact that dye retention may occur in cases in which the serum bilirubin is within the normal limits.

MCGUIRE states that the four most common causes of death in biliary surgery are long-standing jaundice, cardiovascular renal disease, abscess around the cystic duct, and carcinoma. The chief contributory causes are lung complications (including embolus), associated pancreatitis, acute hepatitis, liver stones, and peritonitis. The importance of cardiac complications following operation on the gall bladder is emphasized. Embolic processes in the lung are not unusual.

In cases of gall-bladder stones confined to the gall bladder, McGuire regards cholecystectomy as the operation of choice when there are no unfavorable factors such as jaundice. When stones are present in the common duct and the gall bladder is contracted and not functioning, cholecystectomy with common duct drainage is indicated because the gall bladder is useless for any subsequent procedures.

In cases of common duct stones with an apparently functioning gall bladder, cholecystectomy with drainage of the common duct is a safe procedure if all stones are removed from the common duct. However, complications are so frequent in these cases that probably in most instances cholecystostomy with drainage of the common duct is the operation of choice.

In cases of stones confined to the gall bladder and complicated by jaundice, McGuire usually does a cholecystostomy.

In subacute pancreatitis due to biliary infection, drainage is indicated. In the author's opinion, the infection usually reaches the pancreas by the blood or lymphatic routes. In acute hæmorrhagic pancreatitis ample drainage is important.

Carcinoma of the pancreas and ampulla should not be operated upon. Cholecystostomy is contraindicated when malignancy is found at operation. Either nothing should be done or some form of internal drainage should be established.

In acute hepatitis the following conditions have been found: interstitial hepatitis with jaundice of the liver parenchyma, cholangeitis with biliary cirrhosis, and acute cholangeitis of the smaller intrahepatic ducts.

CAVE reports that from January 1, 1910, to April 1, 1926, a period of sixteen years and three months, there were performed on the Second Surgical Division of the Roosevelt Hospital of New York, 470 cholecystectomies and 105 cholecystostomies. The immediate operative mortality (death in the hospital) in these 575 cases was 6.08 per cent.

Of the 241 patients subsequently traced, 209 had been subjected to cholecystectomy and thirty-two to cholecystostomy.

Of the 209 who had been subjected to cholecystectomy, 182 (86.1 per cent) reported that they were well without symptoms or further operations. Of the remaining twenty-seven, three had been operated upon elsewhere for stones in the common or hepatic ducts, three for stricture of the common duct, and twenty-one complained of digestive disturbances or pain in the right upper quadrant of

the abdomen. No persistent biliary fistulæ were reported.

Of the thirty-two patients who had been subjected to cholecystostomy, eighteen (56 per cent) had had subsequent operations. Eleven had had a cholecystectomy and six both a choledochotomy and cholecystectomy. One had a second cholecystostomy six months after the first drainage operation and a cholecystectomy four months later. Since the last operation, which was performed nine years ago, he has had no abdominal discomfort of any kind.

The most frequent and troublesome complication after cholecystectomy is hæmorrhage. This may be due to an anomalous or friable vessel, injury to the portal vein, or the slipping of a ligature after the operation due indirectly to violent vomiting. Of the thirty-three deaths reported, four were due to hæmorrhage.

Injury to the bile ducts only occasionally causes an immediate complication. If this occurs, a reconstruction operation may be performed at some future time.

Of the immediate deaths in the cases reviewed, four were due to postoperative pneumonia and four to peritonitis.

Cave believes it is best to keep patients with acute gall-bladder inflammation under observation for from twenty-four to thirty-six hours. If by the end of this time there is no abatement of the symptoms, cholecystostomy is the operation of choice.

Three of the immediate deaths in the cases reviewed occurred within forty hours with hyperpyrexia of 106, 107, and 107.2 degrees F. respectively. In cases that end fatally within forty-eight hours the cause of death is probably the absorption of toxins from chemically altered liver cells or infected bile.

The Rowntree-Rosenthal dye test is of value in determining hepatic function and should be used before operation in all cases of disease of the biliary tract in order to determine whether a removal or drainage operation should be done. In the preoperative preparation the use of calcium as suggested by Walters, the forcing of fluids, a high carbohydrate diet, and the administration of glucose by mouth are indicated.

DEAVER and BURDEN state that the surgical mortality of cholecystitis depends largely on the duration of the disease and its complications. The clinical symptoms and the pathological findings often vary. In acute cholecystitis, operation should be deferred until the acute attack has subsided if immediate intervention is not necessitated by perforation or suppuration. Gangrene and acute perforation of the gall bladder are rare. When the latter occurs, a fistula between some portion of the bowel often results.

Calculi are usually the result of cholecystitis. To remove the pathological condition the gall bladder must be removed. In biliary obstruction, the degree of jaundice is dependent upon the extent of the obstruction, the threshold value and the capacity of the kidney to excrete pigment, and the permeability of the blood vessels to pigment.

In cases of jaundice, surgical mortality is probably due more often to failure to establish the bile flow in the presence of hepatic and renal insufficiency than to the loss of blood from oozing. The dangers in obstructive jaundice are the pigment and bile salts in the blood, the tendency to bleed, and liver and kidney insufficiency. The aim in the preoperative preparation should be to combat and control these factors.

In most cases, a diseased gall bladder and a stone in the common duct can be removed safely at one operation. The common duct should be drained with a T-tube.

In acute pancreatitis any abscess or hæmatoma that may be present should be incised and drained. If there is no abscess or hæmatoma, drainage should be placed down to the pancreas. In subacute pancreatitis the patient should be kept under observation and drainage established if a localized abscess forms. Chronic inflammation of the pancreas is usually secondary to inflammation of the biliary tract. Cholecystectomy should be performed to remove the focus of infection.

Stone in the common duct is considered a complication of cholecystitis since nearly all stones are formed in the gall bladder.

In any type of operation on the biliary tract extreme care should be taken that a stone or sand-like material is not overlooked.

The classical syndrome of stone in the common duct consists in colic, jaundice, and chills. In some cases one or two of these symptoms may be absent. The stones should be removed through an incision in the common duct, the duct explored with the finger or a scoop, and a T-tube inserted.

Deaver and Burden do not consider it advisable to delay operation after the diagnosis of stone in the common duct has been made.

The causes of recurrence of symptoms following operation may be grouped as follows:

1. Incomplete primary operation, such as failure to remove the diseased gall bladder, the appendix, and a common duct stone, or to recognize and treat a peptic ulcer and chronic pancreatitis. Cholecystostomy may temporarily relieve the symptoms of cholecystitis, but does not cure and by its mechanical effects may be the cause of new symptoms. Persistence of symptoms after gastro-enterostomy for ulcer may be caused by cholecystitis. Removal of the appendix, even when it is not acutely diseased, is a safeguard against future trouble.

2. Hepatitis and pancreatitis. These conditions may persist for several months after cholecystectomy. On several occasions drainage of the common duct in such cases has given good results.

3. Accidents of the operation, of which injury to the hepatic or common duct is the most serious and unless promptly recognized and treated may result in a stricture with jaundice or a complete biliary fistula.

4. Incorrect diagnosis both before and during operation. The surgeon must be trained in the art of diagnosis and in the recognition of pathological tissue.

BRUCE reports six cases of cholecystitis associated with duodenal ulcer. He mentions the similarity of symptoms and the fact that melæna or hæmatemesis may occur from infection of the gall bladder as well as from ulcer. In the cases reported the prominent symptoms were those of gall-bladder disease.

A thorough examination of the duodenum should be made in all operations for gall-bladder disease.

DOUGLAS discusses the anatomical variations in the extrahepatic biliary system and some of the pathological conditions found. Twelve cases are reported. The various methods of repairing injury of the bile ducts are mentioned.

Injuries to the bile ducts may occur in a simple operation or may be due to pathological conditions rendering the operation difficult, or to congenital abnormalities of the ducts or arteries. The most common causes are traction on the cystic duct, lack of visualization, or blind attempts to stop hæmorrhage.

The site of the injury is usually at the point of union of the cystic and hepatic ducts or the main hepatic duct above this point, or, less commonly, in the common duct.

The symptoms are those of biliary obstruction with or without cholangeitis which at first is usually intermittent but later becomes permanent. More rarely, a persistent biliary fistula is present.

The pathological condition present may show a short, narrow stricture above which small stones, mucus, or biliary détritus is often found. The ducts above the stricture are dilated and the liver is enlarged and soft or, when attacks of cholangeitis have persisted, cirrhotic.

The method of repair must depend on the condition found. Recurrences of symptoms are reported after all methods. Examination of follow-up reports appears to indicate that the best end-results follow suture of the ducts. The next most favorable results follow hepaticoduodenostomy.

Recurrence of symptoms may occur after the patient has been apparently well for months or years and the symptoms may disappear after several months of recurrence.

In two cases reported, the disappearance of late symptoms seemed to be influenced by the administration of bile salts.

CRILE considers the physiological reaction of the brain and liver to be in many ways similar.

"After operations upon either of these organs there is a rapid loss of bodily energy; consciousness fades slowly, and in each case there is little, if any, medication which can influence the unfavorable course . . . . In each, the state of the blood pressure and the circulation give but little clue to the gravity of the condition. The brain and the liver are alike highly sensitive to variations in temperature. Each is a powerful organ manifesting variations within it in a peculiarly dramatic way; each is absolutely essential to life."

About the common duct there is a great sympathetic plexus called by Crile the "abdominal brain." The breaking up of dense adhesions in the search for the common duct causes a generally bodily effect comparable to that produced by manipulation of the spinal cord or of the brain. Experimental trauma of the sympathetic nervous system—interference with the sympathetic nerve supply—produces cytological changes in the liver cells as well as in the brain cells. Strong emotional stimuli cause cytological changes in the cells of the liver. Blocking of the splanchnic nerve supply greatly reduces the systemic effects of manipulation of the viscera.

Clinical experience and experimental research make it clear that, because of these facts, operations upon the common duct require a wide regional block with novocain and, when feasible, a splanchnic block. Also of importance are a clear exposure, a sharp dissection, and the use of a suction apparatus to remove bile or any oozing blood.

The best form of drainage about the common duct is the insertion in the right flank of a drain which terminates in Morrison's pouch. Sudden decompression of the common duct must be avoided.

Another factor of extreme importance is the maintenance of the normal temperature of the liver. This may be done in several ways. In Crile's opinion, the most satisfactory method is diathermy.

JAMES A. H. MAGOUN, M.D.

Corbett, R. S., and Peirce, C. B.: A Clinical Type of Cholelithiasis Resembling Renal Disease; Report of Two Cases. *Surg., Gynec. & Obst.*, 1926, xliii, 459.

The authors describe a type of cholelithiasis suggesting renal disease. While gall-bladder symptoms are not infrequently present in such cases, it is the renal symptoms which bring the patient to the physician. In the two cases reported the renal symptoms disappeared after the gall bladder was removed.

The demonstration of gall stones in the roentgenograms was facilitated by the use of tetra-iodophenolphthalein, not because of absorption by the stone, but because of the contrast afforded by the background of opaque bile. These cases were characterized by the formation of large gall stones, a moderate degree of pathological change in the wall of the gall bladder, and a very fair shadow of the viscus after the oral administration of the dye.

The authors are of the opinion that in both instances the cholecystitis with stones in the gall bladder was the true cause of the symptoms.

JOHN A. WOLFER, M.D.

GYNECOLOGY

UTERUS

Masson, J. C.: Acute Inversion of the Uterus. *Surg. Clin. N. Am.*, 1926, vi, 1329.

Acute inversion of the uterus is a rare complication in labor. In most of the reported cases the condition occurred in a primipara. The predisposing causes are too vigorous compression of the fundus after delivery of the child, traction on the cord, and relaxation of the uterus. Hæmorrhage, the most serious symptom, may be accompanied or immediately followed by extreme shock and pain. The uterus cannot be palpated in the abdomen, and a bleeding globular mass protrudes from the vulva.

The most important considerations in the treatment are: (1) control of the hæmorrhage by hot compresses to the protruding uterus, by pressure, and by the use of pituitrin, (2) control of the shock by transfusion and routine measures, and (3) immediate reduction if the patient's condition will permit it. If reduction cannot be accomplished through the vagina, the abdomen should be opened and the inversion corrected at a later date according to the technique of Haultain or Dobbin.

Whitehouse, B., and Shaw, W. F.: The Causes and Treatment of Uterine Hæmorrhage. *Brit. M. J.*, 1926, ii, 723.

Whitehouse believes that menstruation is the monthly abortion of the decidua of an unfertilized ovum. The menstrual discharge is the lochia of an unfertile abortion. The pre-menstrual endometrium is the menstrual decidua, and its development and life are dependent upon the corpus luteum. Menstrual abortion is initiated by the death of the unfertilized ovum and retrogression of the corpus luteum.

Pathological uterine hæmorrhage falls into one of four clinical groups: (1) epimenorrhœa, (2) menostaxis, (3) menorrhagia, (4) metrostaxis. Epimenorrhœa is the clinical manifestation of hyperactivity of the sex complex. Menostaxis is an incomplete unfertile menstrual abortion. Menorrhagia is the result of uterine insufficiency, which may be developmental, inflammatory, or degenerative. The insufficiency may be associated with lesions in the metrium or the endometrium. Metrostaxis is commonly the reflection of external influences upon the uterus. The accessory factors most commonly associated with irregular uterine bleeding are functional hyperthyroidism and hypersensibility of the sympathetic nervous system. Estimation of the blood tolerance of sugar and of the basal metabolic rate provides important data in the investigation of uterine hæmorrhage at periods of unstable equilibrium, especially puberty and the menopause.

Shaw states that in the normal uterus a definite balance is always maintained between the musculature and the flow of blood through the vessels. This flow can be regulated by contraction of the muscle fibers.

In chronic metritis the muscular tissue is diminished and is separated from the vessels upon which it must act by a mass of inert fibrous tissue. Hence excessive hæmorrhage results.

In subinvolution the uterus remains large and bulky because the muscular tissue is not absorbed and, in addition, the quantity of fibrous and elastic tissue, especially the latter, is increased. It is the collection of this tissue in large quantities around the blood vessels which gives the characteristic microscopic appearance in subinvolution and accounts for the chief symptom of hæmorrhage. The muscular tissue of the uterus is rendered unable satisfactorily to control the flow through the new vessels when a fixed inert mass of elastic tissue is placed between it and the vessel walls. Subinvolution and chronic metritis frequently occur in the same uterus.

In hypertrophy of the uterus the endometrium and the uterine walls are greatly thickened, but there is no alteration in the distribution or amount of the muscular, elastic, or fibrous tissue. Patients with this condition consult the gynecologist in middle life with a history of hæmorrhage and dysmenorrhœa extending over many years. The symptoms are caused by the changes in the endometrium, but the thickening of the wall is due merely to overwork.

Roland S. Cron, M.D.

Pemberton, F. A.: The Relation Between the Treatment of Cancer of the Cervix and the Cell Type. *Am. J. Obst. & Gynec.*, 1926, xii, 536.

The proportion between the three cell types in squamous cancer of the cervix is about the same in the different clinical stages of the disease.

In increasing order of malignancy, the different cell types are the spinal, the adenocarcinoma, the transitional, and the fat spindle.

The type of cell does not indicate whether operation or radium is the better treatment.

The cases having more stroma than cancer tissue respond more favorably to either kind of treatment than those with more cancer tissue than stroma.

In the discussion of this report, Culbertson stated that in his opinion we are not justified in giving a prognosis based on the cell type of uterine carcinoma.

Novak said that there are certain cancers which are definitely of the spinal cell type and others which are just as definitely of the fat spinal cell type, but many cases occur in which the cell type would be differently interpreted by different observers. In

spite of these drawbacks, however, some valuable information seems to have been derived from the study of the cells, particularly the fact that in the cervix, contrary to what might be expected, the spinal cell group possesses a definitely lower grade of malignancy than the basal type.

HEALY stated that 88 per cent of his cases belonged to what might be called the transitional cell or rather favorable group; 18 per cent of these were absolutely favorable, that is, of the adult type of cell, the spindle cell type to which PEMBERTON refers; 70 per cent were of the mixed transitional and spindle cell or adult type, resembling more closely the non-malignant than the highly malignant type of cell; and the remaining 12 per cent were of the highly malignant, undifferentiated, embryonal cell tpye. The treatment of cervical cancer cannot be based in any way upon the histological findings. All that is gained from these is a point of view with regard to the prognosis.

KEENE stated that he is unable to attach any value to the predominating cell as a prognostic index in his clinic.

WARD is of the same opinion as Keene.

SPALDING stated that the unripe cancers of the cervix seem to be the most malignant and have the most rapid growth; when the majority are first seen they have extended to the parametrium or vaginal walls. However, such cancers react better to radium treatment. Spalding's cases of unripe tumors with disease limited to the cervix have been cured. The ripe tumors, although slower in growth and less malignant, seem more resistant to radium; in fact, it seems as if their growth is sometimes accelerated by radium treatment.

E. L. CORNELL, M.D.

ADNEXAL AND PERIUTERINE CONDITIONS

Whitehouse, B.: The Influence of the Corpus Luteum upon Menstruation. *J. Obst. & Gynæc. Brit. Emp.*, 1926, xxxiii, 380.

It is evident from the author's observations that removal of the corpus luteum and therefore presumably also the degeneration of this structure is followed by necrosis of the superficial layer of the endometrium with extensive extravasation of blood into its tissues. Necrosis of the fully developed non-conceptional decidua is associated with the clinical phenomena generally known as menstruation. It follows that this function is to be regarded as an infertile abortion and the voided products of necrosis as the "menstrual lochia." In the production of this abortion, whether it be of the partly mature decidua or of the developing organ, the corpus luteum is a dominant factor.

Ovulation is apparently independent of the corpus luteum, although the actual menstrual period is intimately associated with the loss of the lutein hormone.

The author's observations confirm Shaw's contention that the process of follicle ripening is periodical and responsible for the rhythm of ovarian activity. Follicle rupture, however, is not influenced by any hormone elaborated by the preceding corpus luteum.

On three recent laparotomies for chronic appendicitis in cases in which the pelvic organs were normal the opportunity was taken to aspirate the liquor folliculi from mature follicles and inject the fluid into the blood stream by way of the median basilic vein. The experiments took place during the midpoint of the sexual cycle. The date of the subsequent menstruation was not influenced by the procedure. Amounts varying from ½ to 1 c.cm. of liquor folliculi were injected intravenously without producing any clinical effect whatsoever, either general or local, such as hyperæmia, increased uterine secretion, the production of uterine hæmorrhage, or alterations in the blood pressure.

It was concluded, therefore, that the liquor folliculi in the human species is inert insofar as any influence upon the production of œstrus of the menstrual cycle is concerned. In this respect it appears to differ from the follicular secretion of certain lower animals. The author summarizes his conclusions as follows:

1. Excision or degeneration of the corpus luteum results in necrosis of the endometrium.

2. Ovulation is not influenced by the corpus luteum, but is an index of rhythmic ovarian activity.

3. The liquor folliculi in the human species has no specific function in the sex cycle.

4. Menstruation is the monthly abortion of the developing decidua of an unfertilized ovum, and the menstrual discharge is the "lochia" of this abortion. The pre-menstrual endometrium is the menstrual decidua and is merely a stage in the development of the complete decidua of pregnancy. The premenstrual dilatation of the uterine glands is an artefact produced by the retention of secretion due to constriction of the ducts by the stroma and, later, by extravasated blood.

5. The development and life of the menstrual decidua are dependent upon a hormone elaborated by the corpus luteum.

6 Degeneration of the corpus luteum is normally the result of a "negative phase" produced by the death of the ovum with its corona radiata and the absorption of its products. The cells of the corona radiata are morphologically identical with the large cells of the corpus luteum.

7. The life of the unfertilized human ovum after rupture of the follicle is approximately fourteen days.

8. The rhythmic cycle of events in the human female may be represented as follows: thirteenth day, rupture of the follicle; nineteenth day, development of the corpus luteum completed; twentieth to twenty-seventh day, development of endometrium into menstrual decidua with differentiation into "stratum compactum" and "stratum spongiosum;" twenty-seventh day, death of the unfertilized ovum and the production of a "negative

phase" by the dead cells of the corona radiata; twenty-seventh to twenty-eighth day, beginning degeneration of the corpus luteum followed by necrosis of the menstrual decidua; first to fourth day, continued necrosis of the decidua and removal of the products of abortion by uterine contraction stimulated by pituitary activity; and fifth to twelfth day, regeneration of the endometrium to complete functional activity. CARL H. DAVIS, M.D.

McCready, R. L., and Ryan, E. J.: Roentgenography of the Cavity of the Uterus and Fallopian Tubes, with Special Reference to Its Value in Cases of Sterility. *Am. J. Roentgenol.*, 1926, xvi, 321.

The authors briefly review the development of the roentgenographic study of the female generative organs with the aid of contrast media such as lipiodol. The technique used is described briefly and the difficulties of interpreting the roentgenographic image are discussed. The value of stereoscopic exposures and the necessity of examinations made at varying intervals are emphasized. The article includes numerous roentgenograms illustrating apparent discrepancies revealed by examinations made at different times, and an attempt is made to explain them. These cases are exceptional, however, and the normal patency of tubes is ordinarily demonstrated easily.

One of the chief advantages of the method in sterility due to occlusion of the tubes is that it reveals the point of occlusion, which is a very important factor in determining the indication for operation. In occlusion at the isthmus the prospect of improvement after operation is very poor. The further the occlusion is situated from the uterus, the better the chances for the success of operation.

The authors believe that the results of roentgenography of the tubes can be thoroughly relied upon if the examination is made properly. It has been shown by many cases that not only the diagnosis of patency but also the point of occlusion of a tube can be accurately demonstrated. As practical examples of the efficiency of the test, three cases of sterility are reported briefly.

ADOLPH HARTUNG, M.D.

EXTERNAL GENITALIA

Sherman, E. M., and Norton, S. L.: Further Research in the Problem of Vulvovaginitis in Children. II. Serological Studies. *J. Urol.*, 1926, xvi, 279.

The authors state that by the use of the five preliminary tests for the selection of a suitable complement before the sera from guinea pigs are pooled, the complement-fixation test for gonorrhœa is rendered more reliable. In the study reported, only 70 per cent of the animals tested could be used in the serological tests.

Of the 196 parallel tests with a polyvalent antigen made from ten Torrey strains and a bivalent antigen made from two Torrey strains, 150 (76.5 per cent) gave identical results, thirty-eight (16.4 per cent) showed a stronger reaction with the polyvalent antigen, and eight (4.08 per cent) showed a stronger reaction with the bivalent antigen.

In the parallel tests with active and inactive sera of 105 blood specimens a larger percentage of stronger reactions were obtained with the active sera. However, this was too small a series of tests from which to obtain conclusive deductions. An experimental study of active and inactive sera would be of value in the complement-fixation test for gonorrhœa in which the antibody response is generally weak.

A Kaliski-Breuer control test on all sera gave no evidence of false negatives which could be ascribed to the presence of natural antisheep amboceptor in the patient's serum.

The three courses of provocative vaccine administered did not significantly affect the complement-fixation tests, the clinical signs, or the bacteriological findings.

Children from 4 to 5 years of age with similar clinical pictures gave weakly positive complement-fixation reactions during the period of study. Two children aged 9 and 10 years who gave a weakly positive reaction showed only the latent clinical stage. It was observed that older children with only latent signs gave a negative or only weakly positive serological response.

Children from 1 to 3 years of age in the chronic stage with occasional exacerbations or in the stage with no exacerbations gave negative complement-fixation reactions. Two children in the negative group had reached the age of puberty and had not shown any signs of exacerbation, remaining clinically in the chronic stage while under observation. It is possible that these two children were examples of a spontaneous cure at puberty, a belief held by many observers and frequently expressed in the literature. It has been observed that an exacerbation with increased clinical signs in children over 5 years of age is invariably preceded, accompanied, or followed by a stronger serological response. In babies and very young children, an exacerbation with accompanying increased clinical signs has not produced any increase in the serological response as demonstrated by the complement-fixation test. Therefore it seems that age as well as involvement is an important factor in the production of the gonococcus antibody.

Of the blood specimens obtained for controls from adults with positive histories and clinical signs, 68.8 per cent gave positive complement-fixation reactions and 31.1 per cent gave negative reactions. Of the blood specimens from normal adults, 91.6 per cent gave negative reactions. One evidence of non-specific fixability occurred, four bleedings from one normal adult giving weakly positive reactions with a polyvalent antigen (serum not anticomplementary) and negative results with a bivalent antigen.

None of the seventeen vulvovaginitis strains isolated to date agglutinated with a polyvalent immune serum in dilutions of 1:100 and above.

In five cases the Wassermann reaction was positive. ROLAND S. CRON, M.D.

MISCELLANEOUS

Huggins, R. R.: Ligation of Pelvic Veins in Thrombophlebitis. *Am. J. Obst. & Gynec.*, 1926, xii, 562.

The diagnosis of thrombophlebitis is not difficult. Even when there is doubt, an exploratory laparotomy in no way interferes with the progress of the case. If a mistake is made, the patient will survive and no harm has been done. It is not unusual to discover a small abscess in the uterus or broad ligament which alone will justify the exploration. Such a lesion may be accompanied by chills and a temperature similar to that of phlebitis.

The veins should be ligated through a transperitoneal incision. They need not be excised. If possible, the ligature should be placed distal to the point of infection. If this is impossible, a ligation of the vein somewhat short of the extreme extension of the infection, will probably be beneficial because it will interrupt the blood current and thereby arrest the spread of the infection. The author has not hesitated to ligate both ovarian veins, and in one instance ligated the vena cava. With the exception of a moderate swelling of the legs, which disappears, there are few apparent symptoms from such a procedure. This is true also of ligation of both of the ovarian veins and the common iliacs. In the future, the author will ligate the common iliac in preference to the internal iliac, as this procedure is easier and can be done more quickly.

The mortality in the author's entire series of twelve cases, four of which were reported in 1912, was 33⅓ per cent. Eight cases are reported in detail.

In the discussion of this report, HUMPSTONE stated that he thinks the treatment illogical because of the multiplicity of the veins out of the pelvis. The greatest advance in the management of the condition in the last ten years is its prophylactic treatment by primary repair of all cervical injuries. Humpstone has had considerable success with small repeated transfusions in such cases.

BRETTAUER believes it is not easy to determine which cases should be operated upon.

PETERSON thinks Huggins' suggestion of tying off the pelvic veins for puerperal infection is based upon a wrong conception of the pathology. The main argument against the procedure is that the condition is a blood-stream infection when the damage has been done. It would therefore appear that the tying of veins would do very little, if any, good, and probably would harm a patient who is already badly handicapped by a severe infection.

SCHWARTZ stated that ligation is indicated only in cases which are definitely due to anaerobic streptococci and not due to the ordinary pyogenic organisms, and should be done in such cases only when the lung lesions make their appearance.

POLAK reported that when he has operated in cases with the indications given by Huggins the mortality was higher than in the cases in which he did not operate.

PIPER stated that multiple incisions or any incision where there is no pus may change a condition which is taken care of by the lymphatics into a blood-stream infection.

MILLER believes that the operation described has a definite field, but that it is a very limited one; he cannot agree with Huggins that it should be used routinely in septic thrombophlebitis in the early stages. E. L. CORNELL, M.D.

Sampson, J. A.: Endometriosis of the Sac of a Right Inguinal Hernia Associated with a Pelvic Peritoneal Endometriosis and an Endometrial Cyst of the Ovary. *Am. J. Obst. & Gynec.*, 1926, xii, 459.

Novak, E.: The Significance of Uterine Mucosa in the Fallopian Tube, with a Discussion of the Origin of Aberrant Endometrium. *Am. J. Obst. & Gynec.*, 1926, xii, 484.

Davis, C. H., and Cron, R. S.: A Contribution to the Study of Endometriosis. *Am. J. Obst. & Gynec.*, 1926, xii, 526.

SAMPSON is of the opinion that pelvic peritoneal endometriosis is usually due to the escape of menstrual blood into the peritoneal cavity with a subsequent local reaction. He states that menstrual blood at times passes into the peritoneal cavity as a back flow from the uterus through the tubes, from the tubal mucosa itself, from the perforation of an endometrial hæmatoma of the ovary, and possibly from endometrial tissue on peritoneal surfaces. Menstrual blood, like other irritants, causes granulation and scar tissue formation, adhesions, and peritoneal inclusions. Endometrial tissue which is often found on the surface of these peritoneal lesions or embedded within can be explained only by the assumption that fragments of uterine mucosa in the menstrual blood became implanted at these points or the peritoneum was in some way converted into endometrial tissue by the specific stimulation of some ingredient of this blood.

The experimental work of Jacobson demonstrates that similar peritoneal lesions may be produced in rabbits and monkeys by scattering bits of uterine mucosa in the peritoneal cavity, and clinical observations at least suggest that endometrial tissue may be successfully transplanted in human beings. The local peritoneal reaction toward the menstrual blood creates conditions favoring the retention and engrafting of any living tissue contained in the blood, just as similar reactions make possible the implantation of cancer escaping into the peritoneal cavity.

These implantation-like lesions occur most frequently in the dependent portion of the pelvis and in its normal peritoneal pockets and folds. It is logical to assume that they might occur in a hernial

sac just as tuberculosis and carcinosis have occurred in hernial sacs in cases of peritoneal tuberculosis and carcinosis.

Sampson reports a case of pelvic peritoneal endometriosis associated with an endometrial cyst of the ovary and inguinal hernia. Peritoneal lesions containing endometrial tissue were found in both the anterior and the posterior cul-de-sac and in the walls of the hernial sac. The lumen of the hernial sac had been nearly completely occluded by the endometriosis. Observations made at operation and in the laboratory study of the tissue removed indicate that the pelvic peritoneal lesions and those of the hernial sac had a common origin. From the material escaping into these cavities and from the local reaction, Sampson concluded that the substance responsible was menstrual blood.

Novak reports a histological study of many hundreds of fallopian tubes with particular reference to their contents. In seven tubes, particles of uterine mucosa were demonstrated lying free in the lumen, and in one tube, which contained a pregnancy in its outer third, there was found, between this point and the uterus, a mass of cells which resembled trophoblastic or possibly decidual cells. The nature of endometrial particles, when these were discovered, was indisputable. None of the women from whom these endometrium-containing tubes were removed was menstruating at the time. Five of the seven tubes containing endometrium were removed on the twenty-sixth, fourteenth, eighth, tenth, and ninth days of the cycle respectively. In none of these cases did the endometrium in the tubes show the characteristic picture of endometrium thrown off at menstruation.

In at least five of the cases the particles of free endometrium were so large that it seemed almost impossible for them to have entered the tiny uterine orifice of the tube. The suggestion that they were probably moving toward rather than away from the uterus is strengthened by the finding of definite endometrial tissue in the ovary in at least two of the five cases. The failure to find it in the others may have been due in several instances to the fact that little or no ovarian tissue was removed at the operation and in one case to the difficulty of finding small islands of ovarian endometrioma in a specimen which had been kept in a fixing solution for a considerable time.

Thirteen observations made in the course of operations upon women who were menstruating and whose tubes were open failed to show regurgitation of menstrual blood in a single case. Histological examination of tubes removed during menstruation characteristically showed little or no blood. Moreover, no blood has apparently been observed in the pelvis in the thousands of women operated upon by thousands of surgeons immediately after menstruation although if any of the blood had escaped into the pelvis it could scarcely have been resorbed in such a brief space of time. It seems logical to conclude therefore that while menstrual regurgitation through the tube is possible, it is exceedingly infrequent, too infrequent to explain such a very common lesion as pelvic endometriosis.

Novak believes that endometrial tissue cannot undergo retrograde transportation into the tube. He states that if it takes the smaller ovum at least a number of days to pass down the tube, even with the current at its back, it would probably take much longer for tissue to pass upward against this stream, assuming that this were possible. Moreover it appears incredible that degenerative tissue could thread itself into the tubal orifice, make its way upward, and, after probably many days, still have sufficient vitality to grow where it falls. This seems especially impossible because of the very rapid autolytic and degenerative changes that occur in the tissue cast off during menstruation.

One source of aberrant endometrium may be ectopic differentiation of cœlomic epithelium. This is suggested by the fact that in ovarian endometriosis there may be found all of the stages of the differentiation through which the genital epithelium normally passes, e.g., simple endometrium-like epithelium, with few or no invaginations and little or no stroma; more typical endometrium with or without menstrual reactivity; mucosa resembling that of the tube, etc. The mere presence of hæmorrhage cannot be taken as evidence of menstrual participation of epithelium.

The evidence indicates that if implantation plays a part in the dissemination of the endometrium, as it well may, it is the ovary from which the seed is primarily dropped rather than the tube. All reasons cited by Sampson in favor of the tubal origin of implants speak just as forcibly, and indeed much more forcibly, for the ovary as a primary source. Endometrial tissue from the ovary would theoretically possess much greater vitality than that from the menstruating uterus for it could readily break off from the surface or be cast out in the rupture of hæmatomata without the influence of menstruation.

Davis and Cron report nine cases of endometriosis—one in which the endometrial implants were probably due to transuterine inflation, one of an endometrial implant involving the posterior vault of the vagina, one of endometrial implants on the uterus, and five of chocolate cysts of one or both ovaries.

The menstrual histories show that in an appreciable number of these cases the menstrual periods had been painful from the first. In the determination of the etiology of the condition this fact may be of significance. It may eventually be shown that certain ovaries contain rather large embryonic inclusions of endometrial tissue which cause pain from the first period and eventually result in chocolate cysts, and it is possible that the spill from such cysts might lead to the development of secondary growths in the cul-de-sac or on the uterus. It is possible also that the pain of the first period may be due to a very tight internal os, and that this, associated with a congenital retrodisplacement of the uterus, results

in the escape of endometrial tissue through the tube and subsequent growth in a recent corpus luteum with the later formation of chocolate cysts.

In the discussion of these reports, GOODALL said that endometriosis is a clinical entity which has been established beyond any doubt. It will never be possible from the study of the human anatomy to say that the overflow from the uterus and tubes is or is not the cause or that all of the tissues of this nature arise from pre-existing endometrial tissue in the ovary, but a solution of this problem may come from a study of comparative physiology in lower animals in which there is no menstrual flow. Goodall is inclined to attribute the condition to ectopic embryonal tissue of an endometrial nature in the ovary.

HEANEY reported six cases of endometriosis. In three in which the abdominal wall was involved, the involvement followed a laparotomy. In one case, a case of early pregnancy, examination revealed an adenoma of the rectovaginal septum which looked like a carcinoma and four perforations in the vaginal vault from which hung polypi.

DANFORTH reported that on at least one occasion at operation performed during the menstrual period he saw a small amount of blood come from the tube. Chocolate cysts seem always to be found in association with patent tubes. It therefore has been easier for him to accept the transplantation theory of endometriosis than the metaplastic theory.

SCHWARTZ stated that in the study of endometrial tissue in the ovary the implantation theory has impressed him for two reasons; first, because the surface of the ovary, which is in close proximity to the fimbriated end of the tube, is the most common site of the lesion, and second, because in its earliest stages in the ovary the lesion is always superficially placed.

CURTIS reported that for a year and a half he has been operating, whenever possible, at the time of menstruation. In three cases, menstrual blood was observed coming from the tubes. During the last six months he has encountered approximately fifteen cases of endometrial implants. In nearly all there was a retrodisplacement of the uterus, and in every instance the fimbriated ends of the tubes were patent.

E. L. CORNELL, M.D.

OBSTETRICS

PREGNANCY AND ITS COMPLICATIONS

Thomson, C. J.: The Causation of Still-Birth and Neonatal Death. *J. Obst. & Gynæc. Brit. Emp.*, 1926, xxxiii, 390.

In 100 cases of still-birth and neonatal death reviewed by Thomson the primary causes of death were as follows:

Maternal conditions:	
Eclampsia and albuminuria of pregnancy	6
Syphilis	15
Acute maternal diseases	5
Chronic maternal diseases	5
Hæmorrhage of pregnancy and parturition	6
Complications of labor	20
Unclassified maternal states	2
Fetal conditions:	
Deformities and congenital defects	11
Idiopathic fetal diseases	2
Prematurity	6
Postmaturity	3
Pulmonary lesions	9
Visceral hæmorrhage	1
Placental conditions:	
Extensive red or white infarction	5
Retroplacental hæmatoma	1
Cause not found	5

In the twenty-eight cases of macerated still-born infants the causes of death were:

Maternal conditions:	
Toxæmias of pregnancy	2
Syphilis	10
Acute and chronic maternal diseases	5
Unclassified maternal states	1
Fetal conditions:	
Fetal deformity and idiopathic disease	3
Prematurity	1
Placental conditions:	
Placental insufficiency	3
Cause not found	3

From these figures it is evident that syphilis accounted for over one-third of the deaths in the cases of macerated still-born infants.

In the forty-six cases of still-born infants without maceration the causes of death were:

Maternal conditions:	
Toxæmias of pregnancy	3
Syphilis	2
Acute maternal diseases	1
Unclassified maternal states	1
Hæmorrhage of pregnancy and parturition	5
Complications of labor	18
Fetal conditions:	
Deformities	8
Postmaturity	1
Pulmonary conditions	3
Visceral hæmorrhage	1
Placental conditions:	
Placental insufficiency	1
Cause not found	2

Half of the deaths in this group were due to the complications of labor.

In the twenty-six cases of neonatal deaths, the causes were the following:

Maternal conditions:	
Toxæmias of pregnancy	3
Syphilis	3
Acute maternal diseases	1
Hæmorrhage of pregnancy and parturition	1
Complications of labor	2
Fetal conditions:	
Deformities and defects	2
Postmaturity	1
Prematurity *per se*	5
Pulmonary lesions	6
Placental conditions:	
Placental insufficiency (causing prematurity)	2

Pulmonary lesions and prematurity *per se* were therefore responsible for nearly half of the deaths in these cases.

The best results from preventive treatment are to be expected in the toxæmia and syphilis groups.

CARL H. DAVIS, M.D.

Eden, T. W.: The Indications for the Induction of Abortion. *Brit. M. J.*, 1926, ii, 237.

McIlroy, A. L.: Aspects to Be Considered for the Termination of Pregnancy Before Viability of the Child. *Brit. M. J.*, 1926, ii, 240.

Barris, J. D.: Slow and Rapid Methods of Induction of Abortion. *Brit. M. J.*, 1926, ii, 241.

Price, F. W.: Cardiac Indications for Termination of Pregnancy Before Viability of the Child. *Brit. M. J.*, 1926, ii, 242.

Cole, R. H.: The Plea of Insanity for the Termination of Pregnancy Before Viability of the Child. *Brit. M. J.*, 1926, ii, 244.

Evers, H. H.: The Ethical Aspect of Termination of Pregnancy Before Viability of the Child. *Brit. M. J.*, 1926, ii, 245.

EDEN calls attention to the fact that the medical profession has always recognized that its members should not make themselves responsible for the termination of pregnancy unless there are good reasons for believing that completion of the pregnancy would endanger the mother's life or prejudice her future health. It has never been possible, and probably never will be possible, to lay down strict rules as to the conditions under which abortion may be or should be induced. If it were possible, these rules would need constant revision to bring them into line with progress in the management of disease. It is therefore a matter of practical interest for us to determine from time to time the extent

to which we are agreed upon the conditions which may fairly be regarded as indications for the induction of abortion.

Tuberculosis. There seems to be no escape from the conclusion that, on the whole, pregnancy is a definite risk to the tuberculous mother, and that it is not to the advantage of the community for children to be born with a risk of tuberculosis seven times greater than that in other children. These considerations are very powerful arguments for the termination of pregnancy in women with active tuberculosis.

Nephritis. In women suffering from chronic nephritis pregnancy is always attended by certain very definite risks. There may be a complete breakdown of the renal function during pregnancy, leading to uræmic convulsions, or an intercurrent attack of acute nephritis may supervene. If these complications occur and are survived, the working capacity of the damaged organs will be permanently reduced. In a relatively high percentage of cases of chronic nephritis, accidental hæmorrhage, especially of the toxæmic form which is associated with interstitial hæmorrhage and necrosis of the uterine muscle. Moreover, in addition to the maternal risks, the fetal risks are very heavy; probably not more than 60 per cent of the children survive, many of them dying from prematurity and the effects of transplacental intoxication.

A woman with chronic interstitial nephritis of the azotæmic type may be allowed to continue her first pregnancy provided she is kept under careful supervision. If a serious renal breakdown with uræmic manifestations occurs in this pregnancy, early abortion should be induced in subsequent pregnancies. In the case of any woman with chronic nephritis the occurrence in early pregnancy of albuminuria and œdema of the renal type justifies, if it does not actually require, the induction of abortion.

Bacillus-coli infection of the urinary tract. Bacillus-coli infection of the urinary tract is its acute form, which is not uncommon, does not as a rule lead to serious developments during pregnancy although it is sometimes the cause of an acute fever during the early puerperium which may suggest the onset of puerperal sepsis. Eden saw one case recently in which abortion was necessary.

Diabetes mellitus. Although in the past, diabetes mellitus has been regarded as one of the gravest intercurrent diseases in pregnancy, its risks have been very greatly reduced by treatment with insulin. Simple glycosuria is always amenable to dietetic treatment and never constitutes an indication for abortion.

Valvular disease of the heart. Valvular disease of the heart seldom calls for the induction of abortion. As a rule a young woman with a well-compensated valvular lesion will pass through one or possibly two pregnancies without serious inconvenience. Repeated pregnancies, especially if they follow one another quickly, undoubtedly damage the heart and reduce the patient's expectation of life. The nature of the valvular lesion is of little importance; the chief factor is the state of the cardiac muscle. It is only when signs of failure occur that the condition becomes serious.

Mental conditions. In several cases Eden terminated pregnancy on the advice of a psychiatric physician because the patient had had a previous attack of puerperal insanity or was what is called a "borderline case" and had an unfavorable family history. All of the circumstances of the case should be reviewed by the general practitioner, the psychiatric physician, and the obstetrical surgeon before abortion is decided upon.

Chorea. Chorea is a not very uncommon complication of pregnancy, but is usually amenable to complete rest in bed in a quiet room, careful feeding, and the administration of arsenic. In severe cases with pyrexia, however, the termination of pregnancy may be necessary. In all others, however severe the clonic spasm, the induction of abortion need not be considered.

Toxæmic vomiting. Toxæmic vomiting sometimes proves to be incurable by medical means and must then be arrested by termination of the pregnancy. In London, cases requiring the induction of abortion are extremely rare.

Vesicular mole. Vesicular mole is an invariable indication for the induction of abortion. As a rule, however, an exact diagnosis cannot be made until after considerable hæmorrhage has occurred, the internal os is open, and the finger can detect the vesicular contents of the uterus.

Retention of dead fetus. The retention of a dead early fetus in the uterus is usually diagnosed easily after the elapse of an interval of time sufficient to allow shrinkage of the uterus. In such cases the castor oil and quinine method is usually effective.

Bleeding. The obstetrician should never be in a hurry to interfere with a threatened abortion because it is attended at the onset by severe bleeding. Persistence of bleeding is of more serious import for the life of the ovum than an initial severe hæmorrhage.

McIlroy emphasizes that each case must be treated according to its particular requirements. The obstetrician should advise intervention only after he has consulted with a physician or a clinical pathologist. The induction of abortion is not to be regarded as a substitute for efficient treatment otherwise.

No social or economic interests apart from the medical needs of the case should be taken into account. The obstetrician should not be influenced by any likelihood of defect in the character of the offspring. If completion of the gestation is undesirable for medical reasons, the patient should be urged to avoid subsequent pregnancies. If this course is impossible, sterilization is to be considered. In some cases sterilization is the only course to be adopted.

In discussing methods of inducing abortion, McIlroy states that the slow method is to be preferred,

especially in cases of severe wasting disease such as phthisis in which hæmorrhage has a harmful effect upon the patient's recovery. Abortion can be induced successfully and without sepsis by tents. The dilatation of the cervix with Hegar's dilators and the introduction of a gauze and glycerin drain causes the uterus to empty its contents with little or no hæmorrhage. It must be remembered, however, that the stimulation of the uterus to contract and expel its contents is not always easy. In some cases a small rubber tube or a large catheter inserted into the uterus acts satisfactorily. In cases in which the fetus is palpable, induction may be brought about by passing the rubber tube or by rupturing the membranes and pulling down a leg.

Because of the risk of hæmorrhage and laceration in a patient already devitalized by disease, the rapid method of clearing out the ovum under anæsthesia, as is done in cases of incomplete abortion, is not advisable except in very early pregnancy.

Barris states that before the end of the third month of gestation the uterus can be evacuated readily by the rapid method because, at this stage, the head of the fetus is small and easily collapsible and therefore not much dilatation of the cervical canal is necessary. Moreover, the uterine cavity is not too large for the finger to be able to explore its walls completely and separate the ovum from its attachments.

After the third month of pregnancy the evacuation of the uterus is far more difficult as the fetal head is both larger and harder. In the rare instances in which rapid evacuation of the uterus is necessary after the third month of gestation it is far safer, after having dilated the cervix as much as possible, to incise the anterior fornix transversely, strip up the bladder from the front of the cervix, and then divide the anterior lip of the cervix longitudinally so as to expose the uterine cavity, puncture the membranes, and remove the fetus by grasping a leg. This operation of vaginal hysterotomy makes it possible to empty the uterus completely with no risk of lacerating the maternal tissues, and is to be preferred to an abdominal cæsarean section.

The slower methods are usually indicated also after the third month. Of these, the best consists in the introduction of a small hydrostatic bag through the cervical canal after dilatation of the canal by a laminaria tent or metal dilators.

In cases of vesicular mole the evacuation of the uterus by abdominal cæsarean section has been done with very satisfactory results.

Price emphasizes that the essential cause of cardiac failure lies in the heart muscle. This being the case, the relation to the myocardium of chronic valvular disease and disturbances of the cardiac mechanism can be readily understood.

In a case of chronic valvular disease it is of the utmost importance to ascertain whether, with the valvular lesion, there are coincident changes in the myocardium or blood vessels, or both, and if so, their degree.

In the attempt to determine the prognosis in a given case of chronic valvular disease, the nature and degree of the lesion, its mode of origin, the degree of any existing cardiac failure, the existence of complications, and the risk of sudden death must be considered. It is necessary to determine the extent to which the character of the pulse and blood pressure have been changed and the size of the heart. In cases of aortic incompetence it is necessary to determine the pulse pressure, and in cases of mitral stenosis, the length of the presystolic murmur and the presence and length of a diastolic murmur.

Whenever indications of cardiac failure are noted during pregnancy the obstetrician must consider the degree of the cardiac failure, the stage of the pregnancy at which it supervened, the immediate cause of the attack, and the response of the condition to treatment.

In cases of the first degree of cardiac failure, it is probable, whatever the stage of the pregnancy, that the patient will be able to proceed to full term without risk to life. In cardiac failure of the second degree occurring during the first half of pregnancy—especially during the first three months and without some temporary cause—it is unlikely that the patient will be able to proceed to full term, but efforts should be made to allow her to do so if possible. If cardiac failure of the second degree occurs in the second half of pregnancy it is probable that the patient will proceed to full term. In either case, rest is imperative, possibly for several months.

If severe cardiac failure occurs in the first half of pregnancy, it is exceedingly improbable that the patient will be able to proceed to full term, but even in such cases it is surprising how often the outcome is favorable if the patient is able and willing to rest in bed for several months. If severe cardiac failure occurs in the second half of pregnancy the pregnancy should be allowed to proceed to term if possible.

If extreme cardiac failure occurs at any stage of pregnancy, the pregnancy should undoubtedly be terminated.

Except in extreme cardiac failure, it should be accepted that, as a general rule, the pregnancy should not be terminated until a reasonable period of rest and other therapeutic measures have been tried. If in cases of cardiac failure of the second or third degree proper treatment carried out for a reasonable period is not attended by at least a certain amount of improvement or if a relapse occurs, termination of the pregnancy should be considered more favorably, especially in cases of mitral stenosis.

In any case in which it has been decided to terminate pregnancy an attempt should be made first to treat the cardiac failure in the hope of affording a better chance of a favorable result from the induction of abortion or premature labor.

Cole states that insanity during pregnancy is a somewhat rare condition, constituting less than 1 per cent of the total incidence of insanity in women. While mental disorders of a mild type, such as subacute depression, morbid yearnings, and transient

perversions of one kind or another are not uncommon, they pass over into actual insanity in only exceptional cases.

If insanity develops in the latter months of pregnancy, it usually persists in recoverable cases for some time after the birth of the child, whether the patient is delivered prematurely or not.

Unmarried girls who become pregnant sometimes seek termination of the pregnancy to alleviate their mental distress, in ignorance of the law governing the matter. Cole has seen no such case in which operative intervention would have been justified.

In phobic cases of the obsessional group in which, as the result after consultation, the obstetrician believes that the morbid fear of death as the outcome of the pregnancy is so overpowering as to endanger the patient's life, operative intervention may be indicated.

With regard to cases in which previous insanity has occurred, especially if it occurred after labor, and in borderline cases in which there is an unfavorable family history, Cole feels obliged, unless there are indications presented by the patient's bodily health, to dissent from the view expressed by Eden that labor should be induced before the child is viable. He regards it as doubtful whether the mental disorder would be less liable to occur following premature intervention than after the seventh month or at full term.

Cases have been known in which an insane husband, home on leave from an institution, has caused his wife to become pregnant and the wife has become distracted at the prospect of bearing a child who she thinks may be mentally defective. The fear is a veritable obsession. Cole believes that in such cases there are quite adequate grounds for intervention.

In Cole's experience, the most usual types of insanity occurring in pregnancy are the manic depressive type and dementia præcox. When symptoms of exhaustion ensue in the first months of pregnancy from the severity of a mental disorder, it is doubtful whether the termination of the pregnancy would be beneficial; in fact, it might make matters worse. Refusal of food, insomnia, and suicidal tendencies require most careful observation of the patient, but the pregnancy should be allowed to continue.

EVERS states that no purely speculative excuses for emptying the uterus should be entertained for a moment. The procedure should be most rigidly restricted to conditions in which its value has been proved beyond doubt. These conditions, Evers believes, are extremely few. The burden of proof of indication or justification rests always with those who recommended the operation, and many of the reasons given thus far have been largely speculative and unconvincing.

In conclusion, Evers says that he has been asked by Lyle to give the three following good reasons why the induction of abortion should be practically never considered:

1. The enormous importance that is attached to the value of intra-uterine life today.
2. The great improvement in the medical treatment of most complications which has occurred in recent times.
3. The fact that the dangers originally believed to be associated with pregnancy under certain conditions were most of a speculative nature and groundless.

CARL H. DAVIS, M.D.

Smith, M. G.: On the Interruption of Pregnancy in the Rat by the Injection of Ovarian Follicular Extract. *Bull. Johns Hopkins Hosp.*, Balt., 1926, xxxix, 203.

Within the last few years, considerable study has been directed toward the definite cyclic changes in the vaginal mucosa of rodents during œstrus which can be determined and followed by the examination of vaginal smears. The changes in vaginal smears were first used by Allen and Doisy for testing an ovarian extract. Allen and Doisy have shown that an extract of ovarian follicular fluid will produce in the vaginal mucous membrane of an ovariectomized rat changes which are identical with those seen during the œstrus cycles in normal animals. Smith undertook a series of experiments on rats to determine the effect of ovarian follicular extract upon pregnancy. He draws the following conclusions:

1. It is possible to interrupt pregnancy in the rat by the injection of follicular extract during the first five days of pregnancy.
2. As the pregnancy proceeds from the first to the fifth day the injection of much larger amounts of the follicular extract is necessary to interfere with the course of the pregnancy.
3. From these observations it seems probable that there is at least a difference in function between the follicular secretion and that of the corpus luteum.

HARVEY B. MATTHEWS, M.D.

LABOR AND ITS COMPLICATIONS

Bailey, H.: Trial Labor in the Treatment of 477 Cases of Contracted Pelves. *Am. J. Obst. & Gynec.*, 1926, xii, 550.

In trial labor in 477 cases of contracted pelvis the low-flap cervical section was done when the head did not engage. There were fifty-nine cæsarean sections with no maternal deaths and three infant deaths. Of the twenty-two elective sections, fourteen were low-flap operations. Four patients had a postoperative fever. In three the wound was infected. In two cases in which a vaginal examination was made there was no puerperal morbidity.

Thirty-seven patients were subjected to section following trial labor. Of these, fifty-five were operated upon by the low-flap method. Eleven of the group had been examined vaginally. Twenty-one had a fever of 100.4 degrees F. or higher for two days or longer. In four cases there was a fever for two days, in twelve for less than five days, in one for six days, and in four for eight days. In twenty-eight

cases with a postoperative fever the wound healed by primary union. In two cases it broke down, and in seven a stitch abscess developed. Only one of the patients had a serious postpartum condition. In most of the others the fever occurred in the first few days following the operation. Protection of the incision will eliminate some of the infections as it is probable that they occur when the child and the membranes are brought through the wound. The postoperative period was peculiarly free from the complications that usually follow the classical section.

E. L. CORNELL, M.D.

Banister, J. B.: The Place of Induction of Premature Labor in the Treatment of Contracted Pelvis. *Brit. M. J.*, 1926, ii, 519.

The author states that cæsarean section is being employed too often in the management of pregnancy complicated by contracted pelvis. From his experience in 745 cases of contracted pelvis in which the induction of labor was done he draws the following conclusions:

1. The wider use of induction of labor in cases of contracted pelvis should be urged and is especially necessary today in view of the widespread employment of cæsarean section. It should be urged, furthermore, because it is simple, safe, and unaccompanied by untoward complications and sequelæ.

2. Premature induction of labor is not to be decided upon lightly, but should be dependent upon very careful investigation and frequent examination.

3. It affords the only actual test of the power of the uterus under conditions which imply a reasonable chance of the survival of the baby.

4. It is an attempt to procure what is rightly regarded as the highest aim of the true obstetrician—a natural parturition.

HARVEY B. MATTHEWS, M.D.

Spalding, A. B.: Limitations for the Cæsarean Operation. *Northwest Med.*, 1926, xxv, 526.

Spalding reports on 2,000 consecutive birth records of the Board of Health of San Francisco, California. One thousand, four hundred and eighteen (71 per cent) of the births occurred in hospitals. In 2,020 hospital confinements at the Stanford Hospital in the period from 1918 to 1926, the maternal mortality was 0.6 per cent and the fetal still-birth mortality 2 per cent. In these 2,020 cases, 160 cæsarean sections were performed, one in every twelve cases, with a maternal mortality of 2.4 per cent and a fetal still-birth mortality of 4 per cent. When the operation was done on seriously ill patients there was an increase in the maternal mortality. Of fifty-one patients with eclampsia, fourteen were treated by cæsarean section with a maternal mortality of 28.5 per cent, whereas in the cases of twenty-seven who were delivered spontaneously or by forceps or version, the mortality was 19.9 per cent. The fetal mortality was 28.5 per cent in the cæsarean section group, and 12.2 per cent in the other group.

In the clinic service of the Lane Hospital there were ninety-three cæsarean sections in 3,772 confinements, or one in every forty deliveries. There were six deaths in this group, a maternal mortality of 6.4 per cent as compared with a mortality of 1.2 per cent in cases with other types of operative delivery and a mortality of 0.19 per cent in cases of spontaneous labor. Three of the six deaths in the cases of cæsarean section were associated with eclampsia and one was due to acute miliary tuberculosis. Therefore only two maternal deaths, a mortality of 2.1 per cent, were chargeable to cæsarean section.

The patient with eclampsia is such a poor operative risk that, instead of being benefited by operation, she is distinctly harmed by it.

CHARLES F. DUBOIS, M.D.

Williams, J. W.: Cæsarean Section at the Johns Hopkins Hospital. *Northwest Med.*, 1926, xxv, 519.

The primo indication for cæsarean section is disproportion between the size of the fetal head and the pelvis. All other indications are of secondary importance.

The mortality increases from practically nothing when the operation is performed at the proper time before or just after the onset of labor to 10 or 15 per cent when it is done late in the second stage.

Sterilization by supravaginal amputation of the uterus with burial of the cervical stump is advised to prevent repeated sections on the same patient and to overcome the mechanical effects of uterine myomata obstructing labor. It is indicated also when signs of infection are present. The convalescence following this relatively severe operation is much more satisfactory than that following the simpler classical section because the involuting puerperal uterus does not offer ideal conditions for the combating of infection.

At the Johns Hopkins Hospital, Baltimore, the classical operation is restricted to patients upon whom it can be performed at an appointed time before or at least not later than six hours after the onset of labor. In all other cases, especially those with clinical signs of infection, the supravaginal amputation is done. Since adoption of this plan, only two patients have died from infection following the classical section, and in a series of ninety-five radical sections there was only one death from infection.

In fifty cases in which cæsarean section was done during the past eighteen months, Harris of Williams' clinic made cultures with a sterile cotton swab introduced into the lower uterine segment immediately following the birth of the child and before the fingers entered the wound. He found them uniformly sterile in all elective sections and constantly positive whenever more than six hours had elapsed between the onset of labor and the time of the operation, whether the temperature was elevated or not. In the positive cultures the predominating

bacterium was the streptococcus, which occurred both in the aerobic and anaerobic and the hæmolytic and non-hæmolytic varieties.

Williams agrees with Kronig, Beck, and DeLee as to value of the low cervical section. When the time for the elective section has passed, the low operation gives results which formerly could be obtained only by a radical operation, and sometimes obviates the sacrifice of the uterus.

In 28,000 obstetrical cases there were 363 cæsarean sections, an incidence of 1.3 per cent or one to every seventy-seven admissions. This is extremely conservative considering that one-half of the patients were negroes, in whom the incidence of pelvic abnormalities approaches 40 per cent.

Of 142 sections, 80 per cent were done because of disproportion due to contracted pelvis. The frequent employment of section in placenta prævia, eclampsia, breech and occiput posterior presentation is a serious abuse. CHARLES F. DUBOIS, M.D.

PUERPERIUM AND ITS COMPLICATIONS

Goodman, S. J.: The Treatment of Puerperal Sepsis. *Ohio State M. J.*, 1926, xxii, 849.

The prevention of puerperal sepsis is far more important than its treatment. In the presence of infection the uterine curette is a very dangerous instrument. When obstetrical procedures are carried out with the same judgment and caution as major operations, puerperal sepsis will cease to occur. In the presence of puerperal sepsis, conservatism is the procedure of choice; operative interference is seldom, if ever, justified. Conservative treatment should consist in prolonged and absolute rest, fresh air, good food, and good nursing care. Sera and intravenous injections of chemicals have been disappointing.

In conclusion Goodman says that the present puerperal death rate is a disgrace to the medical profession, but there is little likelihood of any improvement in the statistics until the members of the profession learn to make a correct diagnosis and to recognize the cases in which operative intervention is necessary. HARVEY B. MATTHEWS, M.D.

NEWBORN

Blanco, L. V., and Paperini, H.: Meningeal Hæmorrhage in the Newborn. *J. Am. M. Ass.*, 1926, lxxxvii, 1261.

From the standpoint of the symptoms the authors classify intracranial hæmorrhages of the newborn as extradural, subdural, subarachnoidal, and intracerebral.

The subdural hæmorrhages are the most important, being the most frequent and most to be feared. They occur as the result of forceps delivery and version and as the result of pressure on the head as it traverses the pelvis. They have occurred also in cases of spontaneous and easy birth and in the fetus extracted by cæsarean section. It is possible that tetanic contraction of the uterus, abnormal fragility of the brain capillaries, and infection or toxic diseases are factors.

There are two stages in the symptoms, that of excitement due to the increase of internal pressure, and that of paralysis or paresis due to injury and exhaustion of the nerve centers. The newborn, at first restless, soon becomes apathetic and sleepy.

Asphyxia, the result of meningeal hæmorrhage, is often incorrectly stated to be the cause of many still-births.

Hæmorrhage is the most frequent cause of convulsions in the newborn.

Continual pain is of great importance. The authors state that any newborn child that complains is subject to a grave illness and very frequently this is a meningeal hæmorrhage.

In the cases reviewed by the authors, icterus was a frequent symptom. It appeared between the second and third day. Deluca of Larate's clinic says that icterus in the newborn is usually due to meningeal hæmorrhage produced during birth.

Lumbar puncture in meningeal hæmorrhage ordinarily confirms the existence of such hæmorrhage and is probably the surest of all diagnostic measures. When lumbar puncture yields a uniformly hæmorrhagic, or at least a xanthochromic, fluid without germs, the diagnosis of meningeal hæmorrhage is usually correct. When the puncture itself produces the hæmorrhage, the fluid is not uniformly hæmorrhagic. If the fluid in such cases is centrifugalized, it becomes quite limpid, whereas in meningeal hæmorrhage it remains yellowish (xanthochromic). The centrifugalized fluid from cases of meningeal hæmorrhage shows altered red blood cells or crystals of hæmatic pigment.

The possibility of a complete cure is doubtful, and the prognosis as to life is very unfavorable if the hæmorrhage occurs in the first twenty-four hours. The sequelæ produced include idiocy, imbecility, mental debility, epilepsy, spastic diplegia, and hemiplegia.

The treatment may be medical or surgical. The latter is possible only when the hæmorrhage is localized to cerebral areas having focal diagnostic signs. It varies from simple incision of the cranial suture line to trephining and craniotomy. Medical treatment consists in measures to diminish the cranial pressure and effect hæmostasis. These two objects are both accomplished by spinal puncture, which may be done several times a day if necessary. The intraspinal injection of normal horse serum in doses of from 10 to 30 c.cm. two or three times in twenty-four hours is advised. With the horse serum, the authors have given 10 per cent gelatine solution by subcutaneous injection in doses of from 10 to 20 c.cm. repeated twice in twenty-four hours.

CHARLES F. DU BOIS, M.D.

GENITO-URINARY SURGERY

ADRENAL, KIDNEY, AND URETER

Williamson, C. S.: Further Studies on the Transplantation of the Kidney. *J. Urol.*, 1926, xvi, 231.

Williamson reports a continuation of his studies on experimental transplantation of the kidney. In most of his cases the kidney was placed in a pocket in the neck and its artery and vein were anastomosed to the carotid artery and the external jugular vein respectively. The technique is described in full. In a number of experiments Carrel's method was followed; the kidney was transplanted into the abdomen and circulation re-established through the aorta and vena cava.

All autogenous transplants were made into the cervical area. The excretion of urine was at first slow and periodic, but rapidly increased and within a few hours amounted almost to a small stream. This condition did not last unless the remaining kidney was removed at the time of the transplantation. Williamson found it best to remove the normal kidney between the third and fifth day and thus guard against surgical accident to the transplanted organ. The longest time of survival of the autogenous transplant was nine months. Death was due usually to poor drainage caused by cicatricial contraction of the ureter and overgrowth of the ureteral opening by the skin. In many cases the urine excreted by the transplanted kidney was normal, but it sometimes showed casts and usually contained albumin. The urea content and the rapidity of excretion of phenolsulphonephthalein were normal. The quantity of urine during the first few days exceeded that excreted by the normal kidney.

The transplanted kidney was examined histologically to determine its condition at different stages. During the first eighteen hours there was slight œdema of the tubules with rather marked congestion of the cortex. The tubules and glomeruli were normal, but the latter were somewhat congested. The tubules contained a varying amount of hyaline-like material which the author considered to be coagulated blood serum, although he was unable to account for its presence. There was little change in the histological appearance of the transplant. Failure of function began when hydronephrosis and infection appeared.

For homogenous transplantation the donors were chosen at random and there was slight possibility of breed or blood relationship. In most of the experiments urine was beginning to be discharged at the conclusion of the operation, and during the first day the amount excreted was often as great as, and sometimes greater than, the combined output of both normal kidneys would have been. The specific gravity, however, was often low. The homogenous transplant seemed to function perfectly for from one to ten days. If the normal kidneys were in place, however, the concentration of blood urea was much decreased. The first sign of abnormality, usually noted on about the fourth day, was a slight increase in the size of the kidney. The rate of excretion then began to diminish, this sometimes resulting in the rapid establishment of anuria. If the normal kidneys had been removed the urea nitrogen in the blood increased. Diuretics were then ineffective, and death ensued from uræmia from forty-eight to seventy-two hours after the onset of anuria.

Examination of the transplants was made at intervals. Histological study indicated that the glomeruli first showed abnormality and then the tubules. The first glomerular change consisted in an infiltration with lymphocytes which steadily increased until a few red blood-cells were seen escaping. Later, the predominant changes were in the glomeruli or in the tubules, depending apparently on whether cessation of function was rapid or relatively slow. At the time that the tubules showed infiltration and cellular degeneration an ascending infection appeared. So far as the author has been able to determine, infection does not begin until the physiological changes in the transplant begin to impair and diminish the urinary output.

Only two experiments on the heterogenous transplants were performed. The donors were goats and the recipients dogs. In both cases the recipients died within a few minutes after the release of the circulation through the transplants, apparently from anaphylactic shock. Histological examination showed that the organ was filled with clumped red blood-cells which appeared to be plugging the vessels. There was no evidence of clotting.

The author attributes the survival of the homogenous transplants to the biological reaction of the recipient, and the varying length of life, especially in those reported by Carrel, to a biological relationship between the donor and recipient. Williamson suggests also that the biological reaction of all tissues may or may not correspond to that of the blood.

The author sees a possible surgical application of his study and believes it possible that a healthy kidney removed from a subject recently dead may be successfully transplanted if studies previously made have shown compatibility of the blood.

Fey, B.: The Functional Results of Retention in the Renal Pelvis (Les rétentions pyéliques fonctionelles). *Arch. urol. de la clin. de Necker*, 1926, v, 93.

Retention of urine in the pelvis of the kidney must not be confused with distention of the pelvis. It is

due usually to a functional disturbance of the pyelo-ureteral motor apparatus which controls the evacuation of urine from the renal pelvis to the bladder. The emptying can be studied by pyeloscopy or by fluoroscopy of the renal pelvis filled with an opaque fluid.

Fey and Truchat make a pyeloscopic examination after a functional examination, but never on both sides on the same day. The ureter is catheterized to the pelvis, since, if this is not done, a reflux usually prevents injection of the pelvis. The position of the ureter is determined by the use of an opaque sound. The injection of sodium iodide, which is made with a syringe, is stopped when the pelvic shadow appears distinctly outlined. A plate or a tracing of the image is then made. Gentle injection of a little fluid is done while the sound is slowly withdrawn to reveal any ureteral kink or stricture.

If evacuation is slow, its progress is followed fluoroscopically only every two minutes. The movements noted are indistinct pelvic contractions which, when very accentuated as in certain hyperkinetic pelves, resemble mixing movements, and an evacuation movement. At the ureteral mouth the shadow lengthens and forms a cone-shaped cul-de-sac with its base toward the renal pelvis and its apex toward the ureter. This cone is from 1 to 2 cm. high and at its base is separated from the shadow of the renal pelvis by a furrow. The opaque fluid enters the ureter by rapidly forming a lumbar spindle. The original regular pelvic shadow then returns until the next contraction. The contractions recur at the rate of from two per second to two in three seconds until the fluid is entirely evacuated. Even rapid emptying is not continuous. Each bolus forms at the level of the so-called ureteral bulb.

The evacuation time (from two to eight minutes in normal pelves) is an important criterion of the pelvic motor action. As the time varies with the capacity of the pelvis, the fluid injected is measured. The formation, shape, and rhythm of production and evacuation of the ureteral bulb should be noted. The data obtained in the first few minutes of the examination are generally sufficient to indicate the character of the evacuation and to show the emptying time.

The abnormal conditions caused by pyeloscopy exaggerate but do not create the pelvic movements. Peristalsis originates principally at the ureteropelvic juncture; also, perhaps, in the calyces; and certainly in the ureter.

Normally, the fluid dilates the ureter to the end of the sound, where contractions arise which force the fluid into the bladder. Forcing the fluid across the ureteral neck by increased pressure always causes pain. The renal pelvis normally contains little urine, but has a reservoir function. Disturbance of the function of the sphincter causes a dynamic pelvic retention.

Pelvic retention is diagnosed from the clinical symptoms, the findings of ureteral catheterization, and the findings of a pyeloscopic study of the pelvic motor action. The pathological pelvis shows changes of filling and emptying. A large residue in a dilated pelvis is indicated in the roentgen picture by scattered and movable flakes on a large diffuse shadow with pale outlines. The ureteral bulb formation may be absent or sluggish, or occur only after intervals of many seconds. The bulb form may be irregular and show permanent deformities.

Guyon found in the renal pelvis, as in the bladder, aseptic, septic, complete, incomplete, acute, chronic, temporary, and permanent retentions. Fey classifies retentions as complete or incomplete and with or without distention. Duval and Grégoire distinguish: (1) acute retentions without distention (closed hydronephroses), and (2) retentions occurring in the course of a chronic retention (intermittent hydronephroses). Fey and Truchat call these: (1) acute definite retentions of mechanical origin, and (2) acute transient retentions of spasmodic origin. The latter, the ordinary renal colics, are due to spasms of the pelvic sphincter. The painful crises are symptomatic of a lesion of the excretory apparatus (pelvis, ureter), such as pyelo-ureteral nephroses and the migration of caseous débris, hydatids, stones, or blood clots. They may be produced also during tests of the pelvic capacity and during pyelography by tension on the pelvic walls from slight over-filling. The pain is due to spasm.

A contracted ureter causes attacks analogous to intermittent hydronephrosis. Such acute crises are of a congestive or spasmodic origin. Renal colic has occurred after nephrectomy when a stone was left in the ureteral stump. Distentions principally of a mechanical origin have developed insidiously without producing renal colic. The painful spasms generally occur in undistended pelves. The pain is most severe in small pelves, develops slowly in large pelves, and in large sacs is practically negligible because the fluid flows toward the bladder before the pain occurs.

Whatever the origin of the renal colic, the spasm produces an acute retention in the renal pelvis. If the spasm is transient and mild, the retention is minimal and there is a rapid return to normal. If the spasm is prolonged, the retention reaches or passes the physiological capacity of the renal pelvis and becomes itself a cause of spasm. Thus there is formed a vicious circle which prolongs the painful crisis. Acute pelvic distention from retention occurs, and at the crisis the typical syndrome of intermittent hydronephrosis with tumor and oliguria is seen. Hence prolonged renal colics are today treated by ureteral catheterization. The pain ceases abruptly when the catheter enters the renal pelvis. Prolonged renal colic is evidence of acute retention in the renal pelvis.

The author has never made a pyeloscopic examination at the height of a crisis. In a study of the pelvic motor action between crises valuable information regarding the pelvis and the cause of the spasm may be obtained. In most retentions there is no renal tumor. Such a tumor occurs only

in advanced stages of gross retention with distention.

Chronic complete retention is characterized by absence of contraction. The renal pelvis is filled without pain, and after the withdrawal of the catheter no movement occurs. The shadow form in the roentgen plate remains; no bulb is outlined. Observation for from one-half to one hour shows no emptying. The opaque fluid is gradually diluted by the secreted urine and on the following day the shadow in the roentgen plate is gone. Secondary deformities due to the distention may produce mechanical obstruction. In three cases the author was obliged to recatheterize to empty the fluid.

Chronic incomplete retention may show: (1) slight retardation of emptying (usual in pyelonephritis), (2) marked retardation of emptying with slow formation of the ureteral bulb, (3) a residue, or (4) intermittent retention. In the type with residue the emptying begins normally with distinct contractions, but the boluses form progressively farther apart and finally are apparently stopped, the opaque mass then remaining unchanged for from fifteen to thirty minutes. In other cases the residuum not filling the pelvis persists for some time only at a certain point and may be mistaken for slow emptying.

Intermittent retention occurs in cases of movable kidney with kinking of the ureter.

Retention and distention are distinct, but retention often precedes and leads to distention.

Acute retention often leaves no distention, but always is followed by some excitation of the motor action (exaggerated emptying) or incomplete retention. The pelvis may be dilated or normal, but pyeloscopy constantly shows disturbance of evacuation. Hence the pain is due, not to distention, but to a spasm in a functionally disturbed motor apparatus. Dilated pelves may empty very quickly, and undistended pelves may show retention. The dimensions of the pelvis are difficult to appreciate from the pyelographic image. The capacity of the pelvis varies greatly in the same and different persons. Small deformities of the calyces are unimportant as they depend largely on the incidence of the normal ray and the relation of the calyces to the plate. Pyelographs in series show that the physiological contractions modify the image of the pelvis and calyces. A "T" insertion of the ureter in the pelvis, which is not rare in normal persons, and a swan-neck prolongation of the pelvis noted at the moment of the formation of the ureteral bulb have no pathological significance.

Chronic retention and distention do have a constant relation to each other. Chronic retention without distention is more frequent than distention without retention. Both arise from a change in the pyelo-ureteral muscle. Retention is due to a disturbance of motor action, and distention to a disturbance of motor tone (dilatation from atony). Retention is more painful than distention. The motor action may be the first to return to normal. Marked distention seen in a pyelograph suggests the loss of all pelvic contractility. When dilatation is absent or slight, the most exact information with regard to therapeutic indications and prognosis is furnished by pyeloscopy. Hence the author inquires, not whether there is retention with or without distention, but whether the retention is complete or incomplete and what its degree may be.

Retention in the renal pelvis may be caused by mechanical factors, congenital malformations, dilatation secondary to infection or atony of the ureter and renal pelvis, and spasmodic ureteral obstruction by a hypertrophied circular muscle at the lower end of the ureter. Under certain conditions such as when it is situated at irritative points and in cases of spasmophilia, a calculus or an abnormal artery may excite motor disturbances leading to spasm. There are irritative points which, if repeatedly stimulated, cause a disturbance of the contractility of the renal pelvis leading to chronic retention.

Calculus is an important cause of acute spasmodic retention and seems sufficient to produce all of the disturbances of chronic retention. The retention is generally more marked than the distention. Lithiasis is associated with sclerosis and more or less infection of the renal pelvis.

According to the findings of pyeloscopy, an abnormal artery at the ureteral bulb does not cause mechanical obstruction. The line persists clearly in spite of bulb formation, but the fluid passes well and evacuation occurs normally.

A factor in the development of hydronephrotic crises is an aberrant artery. Such an artery is usually fixed in the ureteral wall by fibrous tissue rich in nerves. Section of the artery stops the crises.

Most movable kidneys have no painful crises; in some, the pelvis is dilated. Movable kidneys empty normally in spite of accentuated ptosis, or empty poorly, or show intermittent incomplete retention due to transitory mechanical obstruction. When the kidney is ptosed the boluses cease to form, the bulb is not outlined, and all pelvic contractions stop. Hence ptosis may lead to retention through dynamic disturbance rather than mechanical obstruction. Kinks and T-shaped junctures of the ureter with the pelvis may prevent emptying.

The author has never studied retention in parietal lesions (strictures) by pyeloscopy because of the fear of causing pain and reflex disturbances of motility. Strictures occur normally after slight contraction of a segment of the ureter and are not pathological unless they are noted repeatedly.

The evolution of pelvic retention is characterized by: (1) a series of painful crises, (2) the intermittent appearance of a tumor with persistent attacks of pain, and (3) the appearance of a fixed tumor with more acute continuous pain, a sensation of heaviness, and a drawing-in of the flank. When the attacks of acute spasmodic retention first occur the dimensions of the pelvis and pelvic evacuation are normal. The fatigue of the pelvic musculature from the recurring attacks then becomes manifested by disturbances of motor action (hyperkinesis followed by

chronic incomplete retention) and by atonicity (distention). The spasmodic attacks then gradually become less frequent and finally cease. With loss of the pelvic motor action, complete retention supervenes and the pelvis becomes passively distended with urine. Hence spasm, an important factor determining hydronephrosis, disturbs the pelvic musculature and the emptying of the pelvis by its duration and repetition and leads to atonic distention.

Pyonephrosis is caused by infection which aggravates pre-existing retention (infected hydronephrosis) or causes retention (pyelonephritis). In the first instance the infection increases the disturbances of pelvic motor function and hastens complete retention. In the second instance it corresponds to an ascending or descending infection of the ureter and renal pelvis. In pyelonephritis the infection leads to retention by causing a functional disturbance of the emptying of the pelvis. The muscles subjacent to an inflamed serous or mucous membrane may be affected functionally. Long persisting infection causes infiltration and thickening of the walls, the formation of kinks, and lengthening of the ureter resulting in an inflammatory stricture across which the purulent urine escapes with difficulty. As ureteral catheterization is usually possible, the mechanical factor seems to be of secondary importance.

The open pyonephrosis is more common than the closed. In these conditions pyeloscopic examinations will show all of the phases of incomplete and complete chronic retention with and without distention. As the relation between the degree of infection and the delay in emptying is almost constant, a vicious circle is established which ends in complete pelvic retention and distention. By pyeloscopy the author has noted that pyelonephritis with good motor emptying is generally cured by the usual therapeutic measures (pelvic lavage, vaccination, etc.) if coli bacilluria does not result from an intestinal condition such as appendicitis, colonic stasis, or helminthiasis, and that when the pelvis empties poorly the usual treatment is often of no benefit.

The therapeutic indications in pelvic retentions are as follows:

ASEPTIC RETENTIONS

1. Acute spasmodic retention without chronic retention is combated by medicinal antispasmodics, suppression of the irritative point, or renal enervation. First, the general condition must be improved. Benzyl benzoate and belladonna are indicated to relieve the crises. If medical measures fail, removal of the irritation point (section of an aberrant artery, removal of a stone, nephropexy) are indicated to relieve the pain. Papin's enervation breaks the reflex arc which causes the spasm. On pyeloscopic examination, Papin noted favorable emptying after this operation.

2. In incomplete retention the irritative point must be eliminated, but whether by antispasmodics or by renal enervation is an open question. When the motor action is satisfactory the retention and distention may regress and the pelvis recover its integrity. Hence measures to free the pyeloureteral region, such as section of an abnormal artery, pyelotomy for the removal of a stone, nephropexy, and the correction of kinks, are indicated. Plastic operations give unsatisfactory results and are associated with the danger of destruction of the principal motor center in the bulb region with loss of the remaining motor efficiency.

3. Complete retention requires nephrectomy. Conservative measures fail.

SEPTIC RETENTIONS

1. Complete retentions, whether associated with infected hydronephrosis or a dilatation pyonephritis, have the same outcome and demand nephrectomy. When possible, the nephrectomy should be primary. In other cases it may be preceded by nephrostomy.

2. Incomplete retentions necessitate treatment of the infection. If the retention is primary, suppression of the cause (stone, abnormal artery, ptosis, etc.) is important. Recovery may be hoped for. The infection disappears either spontaneously or under treatment. If the infection is primary, the condition usually evolves toward pyonephrosis and requires an early nephrostomy before complete retention becomes established. Early nephrostomy probably acts favorably on both the infection and the retention. It places the pelvis at rest, prevents distention, may permit recovery and disinfection, and has a favorable influence on the motor disturbance.

WALTER C. BURKET, M.D.

Beer, E.: Uric Acid and Uratic Stones in the Kidney—Uric Acid Showers and Their Diagnosis. *Surg., Gynec. & Obst.*, 1926, xliii, 436.

Beer calls attention to the difficulty of recognizing uric acid stones in the upper urinary tract in the X-ray plate. Even with the use of the Bucky screen, small stones composed of uric acid may be missed. Beer has had no success in rendering these opaque stones visible by coating them with opaque solutions such as argyrol.

An analysis of 136 stones removed from the kidney during the past four years in Beer's clinic showed that 10 per cent of the stones were composed of almost pure uric acid with only traces of calcium salts. Hence we are running the risk of diagnostic error in approximately 10 per cent of the cases of kidney stone. Beer reports six recent cases in which the diagnosis was made by using all of the usual methods of examination in addition to the demonstration of a filling defect. Usually it was confirmed by obtaining a positive scratch mark on a wax whalebone bougie passed into the suspected kidney.

These studies show that sizable stones, failing to show a shadow on the X-ray plate are not at all infrequent in the upper urinary tract and can be recognized only by the most careful methods of study. Most of the patients with such stones have suffered for a long time. The most valuable objective diagnostic criteria are a diminished output of indigo-

carmine and a defect in the pyelographic filling of the pelvis on the side of the stone.

Beer describes also a group of cases characterized by showers of uric acid which produce a clinical picture similar to that due to stone in the kidney—typical colics on one or both sides associated usually with the appearance of red blood cells in the urine. On standing, the urine deposits crystals of uric acid which adhere to the sides of the container. The freshly voided specimen shows no uric acid crystals. These are precipitated in passing down the ureter, become redissolved in the bladder, and pass out in solution. The mechanism by which this is accomplished is explained by Beer on the basis of the behavior of the reversible colloids of the urine. On several occasions on which specimens obtained by ureteral catheter from one side have shown a precipitation of crystals, specimens obtained from the other side have shown none. In a recent case the urine from the right side was full of these crystals while that from the left side precipitated none. This condition has been found repeatedly.

The diagnosis can be made only by allowing the urine to stand, preferably in a sterile container, for from forty-eight to seventy-two hours. Specimens are sometimes so highly acid that they will remain for months without putrefaction. Occasionally, though rarely, cystoscopy reveals uric acid crystals on the floor of the bladder. In such instances the colloidal reversal had not had time to take place.

It is not claimed that all patients whose urine precipitates uric acid on standing have had recent attacks of ureteral or kidney colic. It is believed, however, that when patients in whom the X-ray, cystoscopic, and pyelographic studies have been negative complain of typical ureteral and kidney colic and their urine precipitates uric acid crystals on standing, there is some connection between the latter phenomenon and the syndrome of which the patient complains. HARRY A. FOWLER, M.D.

Roseno, A.: Postoperative Hæmorrhage Following Nephrotomy and Its Prevention (Die Nachblutung bei Nephrotomien und ihre Verhuetung). 50 *Tag. d. deutsch. Ges. f. Chir.*, Berlin, 1926.

The danger of hæmorrhage after nephrotomy is very great. Hæmorrhage occurs in 9.9 per cent of the cases and in 5.9 per cent of these it is fatal. Combating it by through-and-through suture is not sufficient and injures the parenchyma of the kidney. The blood vessels which are cut in nephrotomy penetrate into the interstices between the papillæ in the parenchyma and in the columns of Bertin where they lie about 1½ cm. apart, and give off branches into the corresponding pyramids.

Where these vessels are cut through, the parts supplied by them are destroyed. A ligature at the point of severance in the Bertin's ligament does not increase the loss of parenchyma. The loss it causes is only a fraction of that produced by the through-and-through suture which is followed by atrophy and infarction.

Therefore in nephrotomy the easily visible vascular lumina in the columns of Bertin which have been cut through should be sought, caught with small forceps, and ligated.

Before such ligations were done in clinical cases, they were done on the kidneys of goats. The author describes the specimens in detail. They show that the healing of the wound was very good, the infarction as compared with that following suture was slight, and the destruction of viable tissue was minimal.

In thirty-three cases in which nephrotomy was performed by the old method in the period from 1913 to 1926, postoperative hæmorrhages occurred in three. In five, a secondary nephrectomy was necessary. Four of the patients died. In the five cases in which the new method of nephrotomy was used there was no hæmorrhage. STETTINER (Z).

Lipshutz, B., and Hoffman, C.: Renal Arterial Variations and Extraperitoneal Abdominal Nephrectomy. *Ann. Surg.*, 1926, lxxxiv, 525.

The frequent variations in the automical types of the renal blood vessels found at autopsy and at operation are of great importance to the surgeon. The manner of development of the kidneys predisposes these organs to anomalies. The renal arteries vary in their number, origin, course, and anatomical relations. Kidney abnormalities are often associated with abnormal blood vessels. There is some variation in the arterial supply in from 20 to 33⅓ per cent of cases.

Two specimens are described in detail. The arteries and veins were unusual in number and origin. The ventral lip of the hilus was absent on both sides. Both specimens showed hypertrophy with moderate ectopia.

In the performance of the usual operation for the removal of a kidney, there are many possibilities for injury. Injury to the parietal peritoneum, the cæcum, and the colon, hæmorrhage from an accessory renal artery or an aberrant vein, and tearing of the vena cava inferior have been reported. These possibilities are practically eliminated by the extraperitoneal abdominal incision. Such an incision is especially valuable for the removal of large cysts or tumors, and its use is associated with less danger of metastasis because the renal vein can first be ligated. Its practical value has been demonstrated also in the treatment of traumatic lesions and horseshoe kidney. CLAUDE D. PICKRELL, M.D.

Joseph, E.: The Removal of Deeply Situated Ureteral Calculi (Entfernung tiefsitzender Uretersteine). 50 *Tag. d. deutsch. Ges. f. Chir.*, Berlin, 1926.

Deeply situated ureteral calculi are often operated upon too late with the result that the kidney must be sacrificed later by secondary extirpation. In the futile hope that the stone which has traveled a distance of 28 cm. will pass the remaining few centimeters, the patient and his physician frequently

delay treatment too long. In the meantime the kidney is destroyed by infectious hydronephritic congestion. Therefore, in cases of deeply situated ureteral stones, the author proceeds as follows:

Medical management is first attempted, with the pushing of fluids, diuretic measures such as the administration of glycerine-lemonade, and the hypophysin injections recently recommended by Bergmann.

If medical measures fail, an attempt is made to remove the stone from the mouth of the ureter by means of the cystoscope. The best method is the introduction of a thin ureteral catheter which can be left in for from twelve to forty-eight hours. The ureteral wall becomes softened and slippery so that eventually the stone may fall through into the bladder or, because of its adherence to the catheter, can be drawn into the bladder. The outlook is still more favorable when two thin catheters can be introduced into the ureteral opening. If it is impossible to introduce a catheter past the stone, it is sometimes possible to insert a whale-bone bougie and allow it to remain in. The author warns against splitting of the ureteral orifice with scissors or the cautery of the operating cystoscope in cases of ureteral stone as the trauma so caused not infrequently leads to complete closure of the ureteral lumen by swelling and produces severe septic phenomena in the kidney. Therefore such measures are not only useless but dangerous. When the calculus is visible in the ureteral orifice, attempts to free it are unnecessary as it will usually free itself.

If operative liberation of an intramural ureteral stone is indicated, the incision usually employed to ligate the iliac artery is used, but instead of making the outer third of this incision, Joseph prolongs it transversely inward across the symphysis. In the lengthened portion, the fascial sheath of the rectus is split and the muscle pulled to one side. The exposure so gained is satisfactory even in very obese patients. The peritoneal cavity is not opened. The midline longitudinal incision recommended by American surgeons and by Key is very convenient and gives easy access to the lower portion of both ureters. For this incision, however, the stone must be firmly fixed so that it will not wander upward toward the kidney when the pelvis is raised.

JOSEPH (Z).

BLADDER, URETHRA, AND PENIS

Colby, F. H.: Bladder Symptoms from Congenital Deformities with Associated Nerve Lesions: Report of Three Cases. *Boston M. & S. J.*, 1926, cxcv, 804.

The author reports three cases presenting urological symptoms due to congenital deformities with associated nerve lesions.

Case 1 was that of a man 37 years of age who, for the past thirteen years, had had difficulty in urination. A herniotomy done during this time was followed by retention, and catheterization resulted in cystitis. Occasional catheterization then became necessary. Four years later an operation for contraction of the internal vesical orifice was done and was followed by considerable relief. A few years later an appendectomy was performed and a small rectal fibroid was removed. Thereafter the bladder symptoms became progressively more severe. During the past year continual catheterization had been necessary. Rectal control was poor and had been growing gradually worse.

Ten years ago the patient was found to have a spina bifida occulta with slight changes in the reflexes in the legs and a small area of diminished sensation over the sacrum. X-ray examination showed the typical bony deformity associated with spina bifida. On exposure of the bony defect at operation performed November 9, 1925, a mass of fat tissue investing a swollen sac of the dura was found. This fat mass was removed and the incision closed. For three or four weeks after the operation the bladder was catheterized at regular intervals. The patient then began to void naturally and he has now no residual urine. Today the rectal symptoms are unchanged and the Achilles reflex is absent, but the numbness about the perineum and the back of the thighs is less marked.

The second case reported was that of a 22-year-old girl who had been an invalid since infancy. Extensive ulcerations made necessary an amputation of the left leg at the thigh and soon thereafter a partial amputation of the right foot. As long as the patient could remember she had had urgent urination and difficulty in the control of the bladder and bowel sphincters. Four days prior to her admission to the hospital she had severe pain in the right lumbar region and retention in the bladder of 500 c.cm. of infected urine. X-ray examination revealed spina bifida occulta with absence of the arches of the fourth and fifth lumbar vertebræ. Because of the severe bladder infection and the bilateral pyelitis, any attempt to repair the underlying defect seemed impossible. Some relief was given by the use of an in-lying catheter and irrigations, but it was insufficient for proper bladder function.

Case 3 was that of a boy of 17 years who, at birth, had an imperforate anus. The anus was operated upon successfully, but the patient had always been in delicate health. He suffered from frequent attacks of pyelitis, and his urine was never free from evidences of infection. X-ray examination showed six lumbar vertebræ, but no coccyx, and a fused kidney on the left side. Operation was not regarded as advisable.

CLAUDE D. HOLMES, M.D.

GENITAL ORGANS

Fronstein, R., and Meschebowski, G.: Recurrence Following Prostatectomy by the Method of Freyer (Ueber Rezidive nach der Freyerschen Prostatektomie). *Ztschr. f. urol. Chir.*, 1926, xx, 222.

In rare cases the disturbances of urination persist after prostatectomy or new ones develop. When this

is due to insufficiency of the musculature of the bladder the condition is a purely functional recurrence. However, there is also the possibility that a correctly performed enucleation of the gland may be followed by a true anatomical recurrence. Adenomatous nodes arise from glandular remains left in the capsule. When renewed disturbance of urination is due to incompleteness of the operation or a carcinomatous degeneration, the condition is a pseudo-recurrence.

True anatomical recurrences are very rare. To prevent them the tumor must be removed *in toto* at the time of the operation, and after its removal a very careful search of the field should be made for nodes that may have been left behind. Recurrences are less frequent following the suprapubic operation than following the perineal operation.

GRAUHAN (Z).

MISCELLANEOUS

Hinman, F., and Redewill, F. H.: Pyelovenous Back Flow. *J. Am. M. Ass.*, 1926, lxxxvii, 1287.

In this article the authors continue their discussion of the condition to which they have applied the term "pyelovenous back flow" and report further facts determined by experiments carried out since the publication of their previous articles. They say: "The most important anatomical facts relative to pyelovenous back flow have to do with the relationship of the pelvis and its branches to the renal venous system. The veins of the kidney, in marked contrast to the arteries, freely anastomose. Particularly in the zone between the cortex and the medulla are rich venous arcades between the larger interlobar veins, many of which are in very intimate relationship to the finer terminal branches of the minor calyces. At some of these terminal pelvic divisions, the veins almost completely encircle the fornix calyces. The venulæ rectæ in the medulla, which are richer, so far as number and size are concerned, as compared to the arteriolæ rectæ, are also in very close relationship to the collecting tubules as well as Henle's loops, and it is this particular area that must be considered with respect to certain aspects of pyelovenous back flow. With respect to the tubular system and its relation to its blood supply, the definite zones of cortex and medulla are differentiated by reason of the renal tubule itself being confined to the cortex. Henle's loop only penetrates to a deeper level, and it is between these two zones in the corticomedullary portion that the venous arcades exist, into which the radiating interlobular veins drain.

"The two important portions of the kidney, as emphasized in the foregoing anatomical discussion, in which the penetration of pelvic contents into the venous system most readily occurs are at the acute angle terminations of minor calyces, where the pelvic mucosa is in intimate relationship to large venous spaces, or in the collecting ducts, also in intimate relationship to rich venules. The difference in appearance of a tubular back flow and a pyelovenous back flow can be demonstrated. The outline of the arcuate veins is characteristic of the latter. Tubular back flow is seldom seen except in the human and dog kidney. Ordinarily, there is a penetration up the collecting ducts for only a short distance.

"The dangers of pyelovenous back flow at the time of pyelography would be removed if the pelvis were first washed out with tenth molar sodium chloride and then with sterile distilled water, thus ionizing the cellular membranes, because the pressure required to produce the back flow will be so much increased that there will be practically no danger of the occurrence of pyelovenous rupture."

The following factors are probably of importance in the occurrence of pyelovenous back flow: diapedesis, osmosis, and permeability of the membranes. The authors state in detail the technique of the experiments by which the existence of this condition is proved. The article includes interesting illustrations.

HENRY L. SANFORD, M.D.

Mixter, C. G.: Urinary Obstructions in Childhood. *Ann. Surg.*, 1926, lxxxiv, 533.

A complete urological examination should be made in every case with symptoms of urinary retention or pyuria. In the author's cases the preliminary urinalysis is followed by tests of renal function, blood chemistry determinations, the making of a cystogram, and cystoscopy with pyelograms and ureterograms. If phimosis and a small external meatus are eliminated, the three usual sites of obstruction are the uretero-pelvic juncture, the vesical insertion of the ureter, and, in the male, the prostatic urethra. A stricture at the uretero-pelvic juncture and faulty implantation of the ureter in the pelvis are causes of hydronephrosis. If nephrectomy is not indicated, a plastic operation may be beneficial. A ureteral kink may be found.

Stricture of the ureteral opening in the bladder may be present with or without cystic dilatation. If cystic dilatation is present, excision of the cyst is indicated. If fulguration is possible, it is the method of choice. If there is no cystic dilatation, slitting of the ureter from the orifice upward for ½ in. may give good results.

Diverticula are rare in children, but may cause obstruction at the vesical neck.

Obstruction in the male urethra may be caused by folds forming valves and by enlargement of the verumontanum. More rarely, a congenital stricture may be present. Two cases of congenital stricture in the prostatic urethra were found due to faulty development. One case was operated upon but the other responded to dilatation. The bladder, ureters, and kidneys showed the typical results of obstruction.

Obstruction of the ureter may be caused by the external pressure of accessory vessels to the lower pole and reduplication of the renal pelvis and ureter. Care should be taken to ascertain if the vessel is

the only source of blood supply to the lower pole. The operative procedure in ureteral duplication depends upon the requirements of the particular case.

Megalo-ureter may be encountered in children and may be bilateral. Its etiology is still in doubt, but the condition is usually considered to be congenital. A careful study may show an obstruction, especially in the prostatic urethra in boys. In the female urethra even valve-like folds may cause an obstruction. Operative procedures in true megalo-ureter have been unsatisfactory because of the patient's poor condition. Slitting of the ureteral orifices may benefit, but bilateral nephrostomy should be considered. CLAUDE D. PICKRELL, M.D.

Hyman, A.: The Clinical Aspects of Surgical Diseases of the Urinary Tract in Children. *J. Urol.*, 1926, xvi, 259.

This article is based on a study of 150 cases of surgical diseases of the urinary tract in children seen at Beer's clinic at the Mt. Sinai Hospital, New York. Attention is called to the increasing consideration which has been given diseases of the urinary tract in infancy and childhood during the last few years. While the early literature abounds in post-mortem reports of such cases, their recognition is now made easy during life by routine examination with small-caliber cystoscopes.

Routine study of these cases was stimulated by Beer in 1907 and since 1911 has been advanced by Hyman, Kretschmer, Hinman, Stevens, Quinby, Lowsley, Helmholz, Bugbee, Thomas and Tanner, and others. Cystoscopes for the work are from 9.5 to 12 cm. long and have a caliber of from 10.5 to 15 F. Intravenous indigocarmine is used regularly as a test for renal function combined with cystoscopy. A regular routine study is carried out exactly as in adults. Young children require narcosis, but in the cases of children from 7 to 10 years of age narcosis is seldom necessary.

The patients whose cases are reviewed by the author ranged in age from 6 weeks to 14 years. The majority were between the ages of 4 and 8 years. Cystoscopy was performed 145 times. Lesions closely paralleling those in adults were found. In 62 per cent of the cases the lesions were in the upper urinary tract. Of ninety-four cases of kidney disease, twenty-one were cases of pyelitis, seven were cases of pyelonephritis, ten were cases of tuberculosis, and twelve were cases of neoplasm. Of forty-eight cases of bladder conditions, twenty-eight were cases of chronic retention. Fifty per cent of the cases were referred on account of persistent pyuria. Hæmaturia in childhood is rather uncommon. Urological diseases of childhood may be masked by intestinal and meningeal symptoms.

Pyuria is the most common sign. In the cases of female children all specimens for microscopic and bacteriological examination must be obtained with the catheter. Very few instances of pyelitis in male children have been seen. Many cases diagnosed as pyelitis without cystoscopy were found to have other more serious complications. The youngest child with pyelitis was a male 6 weeks old in whom jaundice was present for a week.

Tuberculosis is very infrequent in children under 8 years of age. In over 85 per cent of the cases, tubercle bacilli were demonstrated in the smear. Eight of ten cases showed a characteristic cystoscopic picture. In eight cases, nephrectomy was done and followed by rapid improvement.

Hydronephrosis occurred in ten cases, not including twenty in which it was due to obstruction at the vesical neck. In one case, that of a male child 6 weeks old, there was a stricture at the ureteropelvic juncture. Nephrectomy was successfully performed. In six cases classified as congenital there was a stricture at the pelvic outlet.

A perinephritic abscess, cortical abscess, or carbuncle was found in eleven cases. The diagnosis offers the same difficulty as in adults. The usual cause is a staphylococcus infection from a distant focus.

Male children are more often subject to urinary lithiasis than female children. Of a series of 2,000 cases collected from the literature, only seventy-seven were those of females. In from 6 to 10 per cent of the cases, renal calculi are bilateral. Practically all stones originate in the kidney. From 60 to 70 per cent are passed spontaneously into the bladder. In obstinate cases of eneuresis an examination for stone should be made. Rectal examination should never be omitted.

In tumor of the kidney, hæmaturia appears late, and in the cases of children may often fail to occur. It was present in four and absent in eight of the cases reviewed. The first sign in many cases is a large abdominal mass. Most cases are inoperable when they are first seen. In 4 per cent of the cases reviewed the condition was bilateral.

By far the greater number of anomalies of the urinary tract occur in the upper part of the tract. Such anomalies lead to hydronephrosis and pyonephrosis. Congenital malformations in the posterior urethra and the neck of the bladder were found in twenty-eight of the cases reviewed, and a congenital diverticulum was found in three. A marked pyuria in these cases shows how prone they are to infection. Persistent pyuria should suggest the presence of a congenital anomaly.

The etiological factors of chronic retention in children are given by Beer as follows:

1. Mechanical obstruction: extravesical—congenital folds and strictures in the posterior urethra, a pinpoint meatus, a contracted prepuce, new growths; intravesical—contracture of the neck of the bladder, diverticulum, and stones.

2. Neuromuscular: brain disease, spinal cord disease (spina bifida), spasticity of the sphincter without definite neurological signs.

The prognosis is unfavorable, the patient succumbing to renal insufficiency and infection. Diverticula were found in three of the cases reviewed,

all those of young boys, one of whom was only 8 months old. The diagnosis is readily made by cystoscopy. Resection and transplantation of the ureter was necessary. In the series of ninety-four cases of kidney disease, fifty operations were performed with a mortality of 10 per cent. Thirty of these were nephrectomies and the rest pyelotomies, nephrotomies, decapsulations, and exploratory operations.

The author draws the following conclusions:

Diseases of the urinary tract in children are more frequent than we have been led to believe. With few exceptions, the lesions are as varied as in adults. In a large percentage of cases developing pyuria the underlying factor is an anomaly of the urinary tract. The feasibility, safety and practical value of cystoscopy in the young are well demonstrated by the cases reviewed. When present-day methods are properly applied, it is seldom necessary to resort to exploratory operation. The diagnosis of diseases of the urinary tract in children should be made in the cystoscopic room rather than, as happens so often, in the postmortem room. HARRY A. FOWLER, M.D.

MacKenzie, D. W.: Malignant Growths of the Lower Urinary Tract. *Boston M. & S. J.*, 1926, cxcv, 811.

The most important bladder tumors are composed of masses of epithelial cells of one type or another, and are generally known as papillary epitheliomata and papillary carcinomata. These may be benign or malignant. Cases of malignant tumors are characterized by induration, sloughing, and resistance to fulguration. As a rule the malignant tumor is single.

Hæmaturia is an important sign necessitating a thorough investigation. Of a series of 821 cases of hæmaturia, the condition in 70 per cent was due to calculi, tumors, and surgical infections of the kidney. Of 140 cases of malignancy, hæmaturia was the chief complaint in 75 per cent. As vesical carcinoma is a local disease and often remains local for a long time, its rational treatment consists in complete and radical excision. Every effort should be made to bring such cases for early examination and treatment before the involvement becomes general. On account of the ease with which cancer cells grow on denuded surfaces, great care must be exercised in operating on vesical tumors suprapubically not to rub or sponge off the tumor cells.

Cancer cells contain the secret of cancer growth but must have for tumor growth the essential response of the host to furnish stroma and vasculature to permit them to organize and grow. If this response fails, the lesion remains carcinoid or disappears entirely. CLAUDE D. HOLMES, M.D.

Mann, L. T.: The Results of X-Ray Therapy of Malignant Growths of the Urinary Tract. *Surg., Gynec. & Obst.*, 1926, xliii, 529.

The author gives in detail a summary of the results obtained in cases of malignant disease of the urinary tract treated with deep X-ray therapy alone, by surgical procedures and X-ray irradiation, by radium and X-ray irradiation, and by a combination of all three procedures. Some of the cases were treated in the Mt. Sinai Hospital, New York, others at other hospitals, and others in private practice. The technique was essentially the same in all, high-power machines with proper screening being used. The term "course" is used to signify a full erythema dose received at the site of the malignancy.

In the first group of cases, eleven of malignant growths of the kidney, postoperative treatment with the X-ray did not show any better results than were obtained in a similar group of cases in which only surgical treatment was given.

The second group consisted of seventeen cases of carcinoma and two of papilloma. In only three of these was there any relief of the dysuria, hæmaturia, etc. Life was not prolonged, and there were no cures.

The series of prostatic carcinomata treated by deep therapy included twelve cases. In two, the dysuria, hæmaturia, and frequency were relieved for some time, while in the ten others there was only temporary relief or none at all. Life may have been prolonged, but there was no decrease in the size of the tumor mass.

The author draws the following conclusions:

1. The cure of malignant growths of the urinary tract by deep X-ray therapy is a very infrequent occurrence.

2. Alleviation of the symptoms occurs almost as infrequently as a cure and in most cases is only temporary.

3. There seems to be a greater prolongation of life in cases treated by deep X-ray irradiation than in cases not so treated. CLAUDE D. HOLMES, M.D.

SURGERY OF THE BONES, JOINTS, MUSCLES, TENDONS

CONDITIONS OF THE BONES, JOINTS, MUSCLES, TENDONS, ETC.

Krasnobajev, T. P.: The Treatment of Acute Hæmatogenous Infectious Osteomyelitis (Ueber Behandlung der akuten haematogenen infektioesen Osteomyelitis). *Novyj chirurgiesčeskij archiv*, 1925, viii, 354.

At the seventeenth Russian Surgical Congress held in Leningrad in 1925 the author discussed the principles of the treatment of acute osteomyelitis and reviewed the results he had obtained in cases of this condition in the last twenty years.

The treatment should be as conservative and as simple as possible. In the acute initial stage, radical operative measures and general narcosis are contra-indicated. Operation must be limited to incision of the soft tissues to the periosteum under local anæsthesia. In severe septic cases this incision must be made before fluctuation is demonstrable. In mild cases, simple aspiration by puncture may be sufficient. Tamponade is contra-indicated, but occasionally drainage with counter-openings may be established for a short time. Following such treatment more extensive operative measures may be not only unnecessary but actually harmful.

Care must be taken to improve the general condition and to spare the tissues in the operative field. In epiphyseal osteomyelitis complicated by suppurative inflammation of the joints more extensive operative measures are indicated. However, in the cases of small children it may be necessary to restrict the treatment at first to simple aspirations. In cases in which aspiration is insufficient, in severe cases in older children, and in cases in which the pus has broken through the capsule wall and has led to the formation of a phlegmon, arthrotomy should be done. Tamponade is contra-indicated. Resection is indicated in epiphyseal osteomyelitis only in cases of hip disease with gangrene and complete separation of the head of the femur. After the sepsis has subsided, early necrotomy with removal of all visible dead bone must be performed without waiting for the formation of an involucrum. The operative wound must then be sutured. If the proper postoperative care is given, amputation and exarticulation are rarely necessary.

The author reports his experience in 428 cases of acute osteomyelitis in children under 13 years of age. There were ninety-six deaths, a mortality of 22.4 per cent. The highest mortality, 22 per cent, was that of the group of 127 children under 2 years of age.

The osteomyelitis involved the thigh and leg in 148 cases (forty-eight deaths), the head of the femur in ten cases (one death), the ilium in twenty cases (eight deaths), the scapula in seventeen cases (recovery in all), and the epiphyses in 127 cases. Epiphyseal osteomyelitis involved the hip joint in sixty-nine cases (sixteen deaths), the knee joint in thirty-five (two deaths), the shoulder in ten cases (two deaths), the ankle in nine cases (no deaths), the elbow in two cases (one death), and the hand in one case (recovery). In the one case of involvement of the tibial epiphysis without involvement of the knee recovery resulted.

Aspiration of pus by puncture was done in nineteen cases (chiefly cases of hip and knee involvement) with one death. Incision or arthrotomy were done in ninety-one cases, with fifteen deaths. Resection of the hip joint was done in eleven cases, with five deaths.

Of sixty-four cases of epiphyseal osteomyelitis in which bacteriological examinations were made, cocci in chains were found in thirty-six (streptococci in sixteen cases and diplococci in twenty). In this group the mortality was 16 per cent, whereas in twenty-seven cases of staphylococcal infection the mortality was only 10.7 per cent. ALIPOV (Z).

Meyerding, H. W.: Chronic Infectious Arthritis with Multiple Ankylosis of Joints and Complete Disability. *Surg. Clin. N. Am.*, 1926, vi, 1301.

A woman, aged 30 years, had been bedridden for five years with polyarticular arthritis. The left hip, both elbows, both wrists, and the left thumb were ankylosed in deformed positions. Within a period of eleven months, three arthroplasties, a Soutter operation, an osteotomy at the base of the thumb, and a disarticulation of a hammer toe were performed. At the end of that time the patient was able to walk with crutches and braces and to do useful work with her hands.

Seeliger: The Pathological Physiology of the Joints; The Formation of Joint Mice (Zur pathologischen Physiologie der Gelenke; Gelenkmausbildung). 50 *Tag d. deutsche. Ges. f. Chir.*, Berlin, 1926.

In a study to determine the still unknown factor in the etiology of arthritis deformans, the formation of joint mice, and other joint conditions, the author paid particular attention to the synovia. Ninety-seven per cent of the latter is fluid and 3 per cent is solid substances. The latter are in colloidal solution and are mucin-like bodies, cell structures from the inner surfaces of the joint. In inflammatory conditions the cellular content is increased. The reaction is alkaline ($P_H = 8.4$ ions). In cases of arthritis deformans, arthropathies, and free bodies in the joints, the alkalinity is reduced ($P_H = 7.8$ ions),

flakes appear, and the coagulation point is reduced. The author sees in such changes in the synovia one of the causes of the conditions mentioned.

The flaking and the reduction in the alkalinity cause a dry synovitis leading to changes in the cartilage which may advance to necrosis, as described by Axhausen. Traumatic hæmorrhages with the elimination of fibrin also indicate a reduction in the alkalinity and lead secondarily to organic changes.

These theories were proved by the author in experiments on animals. When the synovia was normal, organic substances injected into the joints were absorbed without causing any damage, whereas when the alkalinity was reduced they produced the changes noted in arthritis deformans. Trauma also caused a shifting toward acidity which led to a decrease in resorption and marked changes.

In the discussion of this report BIER stated that he had always emphasized the importance of the synovia but was thinking more of its hormonal than its colloidal action. STETTINER (Z).

Loehr, W.: Ischæmic Contracture (Ischaemische Contractur). 50 *Tag. d. deutsch. Ges. f. Chir.*, Berlin, 1926.

The first stage of ischæmic contracture resembles the cold congestion of Bier which is characterized by swelling, coldness of the skin, bluish discoloration with possibly the formation of vesicles, and more or less marked disturbance of sensation. Primary nerve and vascular injuries are not always present. On the other hand, Loehr always found a marked hæmatoma which, because of its diffuse coagulation in the tissues, could not be moved and therefore, under the tense fascia and skin, and especially under the plica cubitalis which has little elasticity, acted as an obstruction to the circulation. Supracondylar fracture differs from dislocation of the elbow in the fact that in the latter the hæmatoma extends posteriorly and is therefore less apt to cause ischæmic contracture.

In the author's opinion the cause of ischæmic contracture is the obstruction to the outflow of blood caused by a subfascial or subcutaneous hæmatoma. The clinical and anatomical findings in the muscles and nerves are secondary.

In experiments on dogs, rabbits, guinea pigs, rats, and mice it was found that the production of similar conditions resulted in ischæmic contracture with all of the subsequent changes noted in man. Cross sections of the legs of the animals showed that the highly sensitive muscles were necrotic, especially in the center of their muscle bellies, as were also the small nerves and the vascular nerves running in these muscles, but there was no thrombosis. On the other hand, the interfascial connective tissue and the large nerve and vascular branches embedded in it formed a peripheral belt surrounding the muscle bellies which were necrotic in their centers. Accordingly, there was here an active regenerative process. Because of the secondary injury of the vessels and nerves, the replacement of muscle is very gradual.

A guide for the treatment is found in the cause of the condition. When ischæmia impends, the hæmatoma must be removed at once in order to overcome the obstruction to the circulation before the development of paralysis.

Subsequent examinations of a series of patients with supracondylar fracture who were operated upon showed a very good functional result. Even in late cases treatment must not be neglected. All measures should be used which will improve the circulation in the muscle and stimulate the nerves to function. STETTINER (Z).

Hickey, P. M.: X-Ray Clinic on Lesions of the Vertebræ. *Ann. Clin. Med.*, 1926, v, 95.

Hickey calls attention to the fact that roentgen-ray studies of the vertebræ may establish: (1) normal density and normal contour of the vertebræ under consideration; (2) decreased densities indicating a subtraction of the calcium content, which may or may not be accompanied by changes of contour from the usual normal; (3) an increase in density indicating additions to the calcium content of the vertebræ with or without changes in

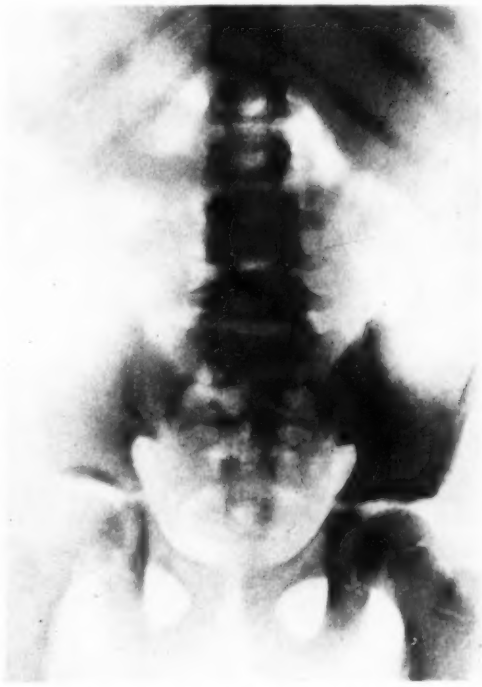


Fig. 1. Roentgenogram of Case 7. Definite union of the third and fourth lumbar vertebræ. Lateral film showed no angulation. History and X-ray findings suggest complete healing of a tuberculous or infectious process.

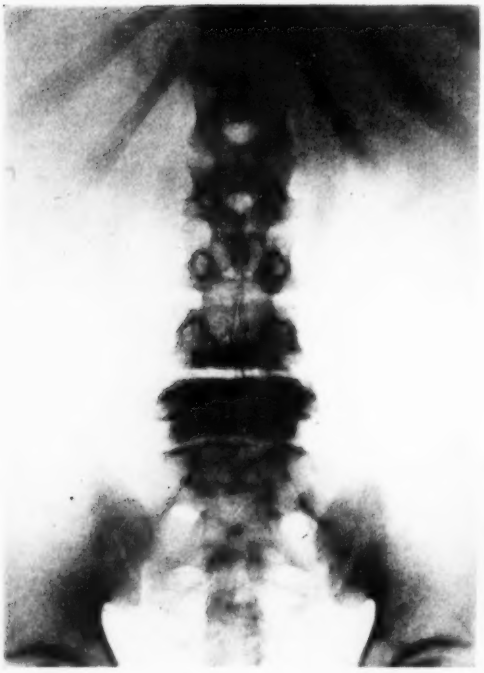


Fig. 2. Roentgenogram of Case 8. Osteomyelitis of the fifth lumbar vertebra secondary to primary infection of the right scapula.

Fig. 3. Roentgenogram of Case 9. Charcot spine with involvement of the second, third, fourth, and fifth lumbar vertebræ. The extra density in the area of the fourth and fifth vertebræ was caused by iodized oil injected into the spinal canal. The painless lesion was discovered when lumbar puncture was attempted.

configuration. All deductions as to the presence or absence of a pathological condition must be based on such findings.

This article reports fourteen cases of lesions of the spine in which a roentgen-ray examination was made and the findings were carefully checked. Most of the case histories are supplemented with roentgenograms.

Cases 1 and 2 illustrate types of compression fractures due to trauma. In the anteroposterior roentgenogram of Case 1 there was superimposition of the shadow of the body of the first lumbar vertebra on the shadow of the body of the second lumbar vertebra with obliteration of the intervertebral space. A lateral film showed a triangular appearance with the apex of the triangle anterior. These findings are characteristic of a compression fracture with an attempt at immobilization through the irritation from displacement. In Case 2, the anteroposterior film did not offer conclusive evidence of the nature of the lesion, but definite evidence of a mild type of compression fracture of the eleventh thoracic vertebra was supplied by the lateral projection.

Cases 3, 4, 5, and 6 were cases of tuberculosis of the spine. In Case 3 the roentgenogram showed coalescence of the seventh and eighth thoracic vertebræ and the shadow of an abscess on either side. In Case 4 the anteroposterior roentgenogram showed definite loss of the intervertebral space between the twelfth thoracic and first lumbar vertebræ, and the lateral projection showed slight involvement of the anterior and upper edge of the second lumbar vertebra, almost complete disappearance of the body of the first lumbar vertebra, and definite destruction of the lower border of the twelfth thoracic vertebra. In Case 5, both the anteroposterior and lateral projections showed involvement of the fifth lumbar vertebra. Case 6 demonstrates the difficulties in drawing conclusions entirely from decreased density. The appearance was that of metastasis rather than tuberculosis although the latter was suggested by the clinical history. A search was made for a primary growth but none was found. The lateral film showed definite destruction of the body of the seventh cervical vertebra and definite thickening of the prevertebral tissues. Operation revealed destruction of the seventh cervical vertebra with definite caseous material.

In Case 7 the anteroposterior and lateral projections showed definite fusion of the third and fourth lumbar vertebræ and the lateral view showed no angulation. Five years previously the patient had what was thought to be Pott's disease, but it may have been an infectious arthritis.

Case 8 was a case of osteomyelitis of the fifth lumbar vertebra secondary to osteomyelitis of the right scapula. The anteroposterior projection showed a decided change in the contour and density of the fifth lumbar vertebra and loss of the intervertebral space between the fourth and fifth lumbar vertebræ.

Case 9 was a case of Charcot spine. There were no symptoms referable to the spine, but Hickey has noted that in tabes the X-ray examination frequently reveals structural changes in the spine which were not suspected from the clinical history. The destructive lesion involved the second to the fifth lumbar vertebræ. There were areas of increased and of decreased density and a definite change in contour.

Case 10 was a case of osteoclastic metastasis of carcinoma of the breast. The roentgenograms showed a decrease in density of the eighth and twelfth thoracic vertebræ and apparent telescoping of the vertebral bodies.

Cases 11 and 12 were cases of the osteoblastic type of metastasis of carcinoma of the prostate. In Case 11 the malignancy was first manifested late in life and the involvement of the spine was limited to the third lumbar vertebra. In Case 12 the condition began at the age of 44 years and the spinal metastases involved all of the lumbar vertebræ and the pelvic bones. There was an irregular mottling indicating subtraction and addition of lime content. This is typical of the osteoblastic type of carcinoma secondary to carcinoma of the prostate.

Cases 13 and 14 were cases of multiple myeloma. The roentgenograms showed the multiple circular areas of lessened density which are pathognomonic of the condition. CHARLES H. HEACOCK, M.D.

Konjetzny: A Contribution to Our Knowledge of Perthes' and Koehler's Disease (Zur Kenntnis der Pertheschen und Koehlerschen Krankheit). 50 *Tag. d. deutsch. Ges. f. Chir.*, Berlin, 1926.

Following a study of fifteen cases of Koehler's disease, two cases of Perthes' disease, eight cases of necrosis of the semilunar bone, and numerous cases of so-called osteochondritis of the knee and elbow, the author agrees with Axhausen that in the group of diseases mentioned we are dealing with a subchondral epiphyseal necrosis with little or no destruction of cartilage. He demonstrated this in the roentgenogram and specimen obtained in a case of Perthes' disease in a 17-year-old boy in which the head of the bone was resected. Along the anterior saw cut there was an extensive subchondral necrosis resembling an infarction. The microscopic picture also showed complete bone and marrow necrosis with a well-defined margin.

The author has seen similar more or less wedge-shaped subchondral necroses of the epiphyseal bone and marrow also in Koehler's disease. Axhausen has called attention to the fact that, under favorable conditions, a gradual replacement of the necrotic tissue by the living surrounding tissues may occur and result in ideal healing. The author observed this in the case of an 11-year-old boy with disease of the first metatarsal which was treated by the application of supporting bandages and was regularly controlled by X-ray examinations. In this case resection of the head of the bone was not done, but a small section 5 mm. thick was trimmed off parallel with the axis of the metatarsal. The author attributes to this method the ideal regeneration of the severely diseased metatarsal head and believes that the necrotic tissue and segment of the head which is contiguous to the normal portion of the epiphysis and the femoral neck should be removed also in Perthes' disease.

Konjetzny was able to study the outcome of severe Perthes' disease in the case of a patient who was under observation for eight years and ultimately died of tuberculosis. This case demonstrated the gradual healing of the disease under conservative management, but shows also that even after eight years complete healing had not occurred, since, surrounded by exuberant masses of cartilage, necrotic bone fragments were found in the form of sequestra although the major portion of the extensive epiphyseal bone necrosis which was visible in the first X-ray plates had become absorbed and replaced. Such residual necroses, which are observed frequently also in old cases of Koehler's disease, may be a source of continuous irritation leading ultimately to a typical secondary arthritis deformans. The beginning of the latter condition was noted in some of the author's specimens.

In conclusion the author discusses the question as to whether the operative treatment mentioned may not hinder the formation of such inclusions of dead bone in the exuberant cartilage so that truly complete healing may occur under otherwise conservative treatment. STETTINER (Z).

Juengling, O.: The Results of the Roentgen Treatment of Bone and Joint Tuberculosis in the Region of the Foot (Ergebnisse der Roentgenbehandlung der Knochen- und Gelenktuberkulose im Bereich des Fusses). *Acta radiol.*, 1926, vii, 14.

In the surgical clinic at Tuebingen, sixty-four cases of tuberculosis of the foot were treated with the X-rays during the period from 1917 to 1924. In thirty-six the condition involved the tarsal joint, in twenty-one the midtarsal bones, and in seven the toe joints. Twenty-four cases were of the spongy type, and in forty there were sinuses. Thirty-five of the sixty-four patients were children.

According to the dosage used, the cases may be divided into two groups. One group was treated before the autumn of 1919 with strong doses, and the other group was treated since then with weak

doses. The first group included thirteen cases, seven of which were children, and the second group included fifty-one cases, twenty-eight of which were children. The results are shown in the table. The numbers in parentheses indicate the cases in children.

Cases Treated Before 1919 with Strong Doses

	Non-active	Benefited	Stationary	Aggravated	Total
Midtarsals...	4 (3)	.....	....	..	4 (3)
Tarsal joint..	3 (2)	.....	3 (1)	..	9 (4)
Cases Treated Since 1919 with Weak Doses					
Toe joints...	6 (3)	1 (1)	....	..	7 (4)
Midtarsals...	14 (8)	2 (2)	1 (0)	..	17 (10)
Tarsal joint..	15 (9)	10 (5)	2 (0)	..	27 (14)

The prognosis of tuberculosis of the toe joints and the midtarsals treated with the X-rays may be considered favorable and as good in adults as in children and in closed conditions.

More than half (55.5 per cent) of the cases of tuberculosis of the tarsal joint could be transferred to the non-active group. The fistulous cases that went on to the non-active stage were much more numerous among the young subjects than among the adults, whereas the cases of the spongy type which showed similar improvement were equal in number among the adults and young subjects.

The results obtained from treatment with weaker doses were far better than those obtained by the strong doses. In nine cases treated with strong doses there were three recurrences and four amputations. In the group of twenty-seven cases treated with weak doses there was practically no instance of lighting-up of the infection and amputation was necessary in only two.

Abscesses should be opened. The injection of iodoform does not seem to have any bearing on the prognosis. Attention to the general health, fresh air, sunshine, and nourishing food is of importance.

The X-ray treatment of tuberculosis of the joints of the foot should never be undertaken without simultaneous orthopedic treatment. As ambulatory treatment the author recommends the application of plaster-of-Paris bandages or the use of some apparatus that will take the weight from the affected joint. The apparatus must be used even after the condition has reached the latent stage.

It is advisable to try radiation in every case of tuberculosis of the foot joints. In serious fistulous cases in laborers, however, care must be taken not to delay operative interference too long when two treatments by irradiation are not followed by definite improvement.

SURGERY OF THE BONES, JOINTS, MUSCLES, TENDONS, ETC.

Payr: Plastic Operations on the Large Ball and Socket Joints (Plastik an den grossen Kugelgelenken). 50 *Tag. d. deutsch. Ges. f. Chir.*, Berlin, 1926.

Payr discusses plastic operations on ball and socket joints, the myodynamic demands of which are much more difficult to meet than those of the more fixed joints as they require an interplay of muscles in three axes.

In the case of the shoulder it is usually necessary, because of the unsatisfactory results of plastic operations, to be satisfied with movement of the entire shoulder girdle, but in the position of adduction this is not sufficient. Accordingly, a successful plastic operation is desirable especially for laborers and young persons. When the loss of substance and muscle defects are extensive the difficulties are insurmountable. Resection of the head of the humerus leaves a shoulder joint without sufficient accuracy of motion or direction.

The insertions of the scapular muscles endowed with little power of rotation are crowded together in one small area. Therefore, in ankylosis, adhesions are formed with them, marked induration occurs at certain points, the bursæ become obliterated, and the subscapularis becomes contracted.

A good exposure without permanent injury of the muscles is difficult. In mild cases, the "half Langenbeck" with chiseling off of the major tuberosity is sufficient and permits bloodless stretching of the subscapularis. When the ankylosis is broken up from above, the osteoplastic liberation of the deltoid origins from the clavicle and scapula makes it possible to preserve all of the muscles attached to the tuberosity. These as well as the newly formed head must be covered by the fascia. Early passive motion in all three axes is necessary for a successful result. When a plastic operation is contra-indicated in rigidity of the arm in adduction, arthrolysis or a wedge osteotomy at the surgical neck followed by support of the arm should be done. The author reports a case in which a very excellent result was obtained by a plastic operation.

In the hip, the outlook as regards both movement in general and movement in the joint is more favorable. The removal of the greater trochanter is better than the liberation of the muscle insertions and opens up the joint like a key. The points at which the vessels enter the trochanteric fossa must be preserved for the sake of the blood supply of the neck and head of the femur. A plastic operation on the joint and a pseudarthrosis in the neck of the femur near the joint are antagonistic. In osteoplastic hypertrophic conditions in which the femoral neck is club shaped, it is best to perform a saddle osteotomy at the neck of the femur and cover it with fascia, whereas when there is a slender fixed head the plastic operation is best. With proper instruments a very good head and acetabulum may be refashioned.

In cases of marked atrophy of the bone, pseudarthrosis is more apt to be successful even though only flexion and extension are regained. The plastic operation on a ball and socket joint demands, in addition to bandaging of the limb in abduction for from four to six weeks, early movement in three axes and measures to prevent adduction and flexion contracture. False positions are difficult to correct later.

Some joints subjected to a plastic operation are transformed by absorption of the head into saddle joints, whereas others retain a good form. Well-functioning pseudarthroses with free flexion and extension are often without a Trendelenburg sign, but the limping is not always due to insufficiency of the small gluteal muscles, being caused also by flexion and adduction contractures. In bilateral ankylosis the right side should be operated upon first.

Of thirty-five ankyloses of the hip, nine of which were fibrous, a very good or good result was obtained in 66 per cent. There were two deaths (one due to fat embolism and the other to coronary sclerosis), in twenty plastics, and thirteen saddle pseudarthroses.

In the discussion of this report, SCHANZ (Dresden) stated that he has mobilized a large number of ankyloses in a similar manner (interposition of a fat pad), but the heads of the bones wore off and he regards it as doubtful whether the modeling of a new head is worth while. As the results of subtrochanteric osteotomy in hip-joint ankylosis are good, he performs this operation in a large number of cases.

WULLSTEIN (Essen) reported that he has often exposed the shoulder joint by Payr's method and finds that the procedure gives good exposure. In the hip, he has effected a saddle pseudarthrosis, which has been done frequently also by Payr. In Wullstein's opinion, the cause of resorption of the head is generally to be sought in injury to the blood vessels. STETTINER (Z).

Whitman, A.: Remarks Prefatory to a Cinematographic Presentation of Late Results of the Reconstruction Operation. *J. Bone & Joint Surg.*, 1926, viii, 803.

The reconstruction operation on the hip is an operation of necessity rather than of election as it is usually done as a last resort. The two chief indications for which it is performed are ununited fracture of the neck of the femur and arthritis deformans of the hip. The results to be achieved are relief of pain, stability of the joint, and mobility of the joint.

The reconstruction operation is a more certain procedure than bone grafting or pegging because, in patients of the class subjected to it, the power of bone regeneration which is so essential to bone grafts is usually very low.

In arthritis deformans the aim is to relieve the pain by shaving off the cartilage and margin of the femoral head, thus reducing the size of the head so that friction with the acetabulum is diminished. Transplanting the trochanter downward in such cases tends to prevent the troublesome flexion-adduction deformity.

An incision in the shape of a half U is begun an inch below and behind the anterior superior spine and continued across the femur at a point about 3 in. below the apex of the trochanter. The base of the trochanter is then separated from the shaft with a wide chisel and turned upward with its muscle attachments. The capsule is opened and the greater part of the head is removed with a curved chisel. In order to excise all of the diseased bone it may be necessary to remove all of the head. The remaining stump is smoothed off carefully with a file but not covered with membrane. A thin section of bone is shaved off the shaft just below the site of the trochanter and the trochanter pulled down and fastened to this freshened shaft with deep sutures or a bone screw. A spica is then applied with the thigh extended and abducted. This is left on for several weeks.

Of greatest importance in the after-treatment is the maintenance of the limb in abduction and hyperextension in order to overcome the tendency toward the flexion-adduction deformity.

From 1916 to date about eighty reconstruction operations have been done at the Hospital for Ruptured and Crippled, New York. Of the first forty patients subjected to it, twelve reported back for follow-up study. Four who were operated upon for ununited fracture showed an average flexion of 44 degrees, abduction of 10 degrees, and 1 in. of shortening. Five who were treated for arthritis deformans had an average flexion of 46 degrees, abduction of 11 degrees, and ½ in. of shortening. Both groups were free from pain, and all but one had returned to their normal activities.

Most of the surgeons discussing this report stated that they preferred the reconstruction operation to bone grafting or arthrodesis.

The article contains twenty-five cuts showing the results of the procedure. WILLIAM A. CLARK, M.D.

Abbott, L. C.: The Correction of Deformity in Quiescent Disease of the Hip. *J. Am. M. Ass.*, 1926, lxxxvii, 1095.

Abbott describes the methods used for the correction of deformity in quiescent disease of the hip at the Shriners' Hospital for Crippled Children, St. Louis. The cases are divided into two groups, those in which the deformity was corrected without ankylosis of the hip and those in which it was corrected with ankylosis. Three cases of each type are reported.

In the first group the deformity was corrected and the shortening reduced by division of the contracted soft parts followed by skeletal traction for several weeks until the maximum correction was obtained. Arthrodesis of the hip was then done by cutting a notch between the upper surface of the remaining portion of the neck and the inner surface of the great trochanter, accurately fitting this denuded area against the upper part of the acetabulum and the adjacent wing of the ilium, and reinforcing the area of contact with bone chips.

In the group of cases in which the deformity was corrected with bony ankylosis the joint was ankylosed in flexion, abduction, and external rotation. To avoid the complications of the usual subtrochanteric osteotomy of the femur in such cases, a new method was employed which combined a transverse subtrochanteric osteotomy with gradual

correction of the deformity to allow the fragments to become embedded in soft callus. When this was done, the gradual bending at the site of deformity could be done without the risk of displacing the fragments. PAUL C. COLONNA, M.D.

FRACTURES AND DISLOCATIONS

Henderson, M. S.: The Cause and Treatment of Ununited Fractures. *South. M. J.*, 1926, xix, 746.

It is difficult to assign a definite cause for delayed union or non-union in any given case of ununited fracture. One or more factors may be responsible, such as devitalizing trauma, the interposition of muscle or fascia with extensive overriding, inadequate reduction, improper apposition of fragments, interference with the blood supply, and too early weight bearing or resumption of function.

The work of Robison and his coworkers appears to have sufficient basis on which to build a reasonable hypothesis to explain the apparent interference with the normal processes of ossification. This theory is based on the presence in the osteoblasts and hypertrophic cartilage cells of an enzyme (phosphoric esterase) which acts on the phosphoric esters of the blood. It has been shown that amino acids such as would be formed by the autolysis of dead tissue or hæmatomata decalcify the bone appreciably and thus tend to reduce the amount of the enzyme present. The enzyme is inhibited in its action by an acid medium. The fact that this unfavorable influence on ossification does not occur in all cases of fracture is not proof that it cannot happen in some.

If this careful experimental work can be accepted, fractures produced by severe injuries with consequent serious trauma to the soft parts should be regarded with suspicion. It is reasonable to argue that such fracture should be opened and cleaned of the damaged tissue and hæmatomata, accurate reduction should be obtained, and a dry field secured.

The massive bone graft is the method of choice in cases of non-union and leads to a higher percentage of cures than any other method. In delayed union, good results are obtained in a large percentage of cases by any method which exposes the fragments, realigns them, and maintains good apposition.

Edwards, H. C.: The Mechanism and Treatment of Backfire Fracture. *J. Bone & Joint Surg.*, 1926, viii, 701.

This report is based on forty-two cases of backfire fracture which have been treated at the Kings College Hospital, London, since its institution in 1920.

Backfire fractures may be divided into two groups, the direct, which result when the starting handle of the automobile flies out of the hand and strikes the forearm, and the indirect, which occur when the handle does not leave the hand. The fractures are of the following types:

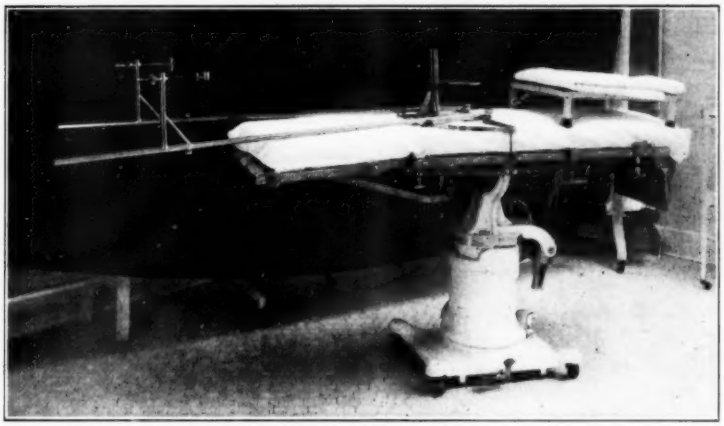
1. Fracture through the base of the styloid process of the radius, with or without involvement of the styloid of the ulna. This may be either an abduction or an adduction fracture.
2. Colles fracture, due usually to indirect violence but occasionally caused by direct force.
3. High Colles fracture, due probably to direct violence and occurring from 1½ to 2¾ in. above the lower end of the radius.
4. High fracture through the radial shaft at the juncture of the middle and lower thirds, due almost certainly in all cases to indirect violence.
5. Fracture of the styloid process of the radius and the inner and posterior margin of the radial articular surface.
6. Separation of the radial epiphysis.
7. Mixed fracture involving the carpal and metacarpal bones.

In the treatment of a Colles fracture at the Kings College Hospital reduction is often effected by manipulation under anæsthesia by the method of Sir Robert Jones, care being taken to increase the deformity before attempting to restore the fragments to the correct alignment. Particular attention is paid to the correction of the backward tilt of the lower fragment. After the reduction, a Carr splint is applied. Edwards considers the Carr splint most satisfactory as it is cheap, easily made, does not hamper the movement of the fingers, and, being wooden, permits X-ray examination. The palmar flexion is adjusted according to the requirements of the particular case. Massage and active and passive movements are begun after the callus uniting the fragments has become sufficiently firm. In the average case this usually requires at least fourteen days.

Open operation is indicated in these cases when several attempts to reduce the fracture by manipulation have failed and when, in old fractures, the wrist is weakened by an uncorrected backward tilt. If the callus between the fragments is soft, it is cut through. Any irregularities likely to prevent reduction are then removed and the fragments are brought together in good position.

If the fracture is old and firm bony union has taken place, an osteotomy through the malunited fracture is necessary. In the procedure which has been found most satisfactory, the bone at the site of the fracture is cut through with a curved gouge and the surfaces of the fragments are so fashioned that the upper surface is convex and the lower surface concave, the one fitting accurately with the other. In this way a false ball and socket joint is made. The distal fragment is then rotated forward so that the backward displacement is eliminated and the radial articular surface faces forward at right angles to the long axis of the radius. No plate is necessary. The periosteum is stitched over the fracture and the skin incision closed. The forearm is then put up in the corrected position on a Carr splint.

In fracture high up on the radial shaft the lower fragment is carried backward with the carpus and



Clark.—A Light Portable Extension Frame.

the extreme upper lip of the lower fragment may be displaced in front of the proximal fragment. On account of the size of the fragment, the correction of the backward displacement is readily effected under anæsthesia. A Carr splint or a posterior angular and a short anterior splint are then applied. Open operation is necessary when there is a marked and irreducible overlap and in old cases in which the backward tilt is uncorrected and will not yield to manipulative methods. In such cases the use of a Lane plate may be necessary. After the operation a Carr splint or a posterior angular and a short anterior splint extending from the wrist to the bend of the elbow are applied.

In fractures through the lower epiphysis, reduction is attempted as for a Colles fracture. Because of the danger of displacing the lower fragment early massage and movement are contra-indicated until satisfactory union has taken place. As soon as the swelling attendant upon the fracture has subsided, a plaster splint is used. In the cases of young adults the plaster is removed and massage and movement are begun after three weeks. In the case of a child the arm is allowed to go free after immobilization for a month. If reduction cannot be effected by manipulation, operation is performed. The use of a plate is to be avoided in these cases. A Carr splint is applied and replaced by plaster-of-Paris when the sutures are out. RUDOLPH S. REICH, M.D.

Clark, W. A.: A Light Portable Extension Frame. *J. Bone & Joint Surg.*, 1926, viii, 750.

Clark describes a frame for obtaining extension of the leg in the reduction of fractures or the application of a cast for any hip or leg trouble. This frame was designed for use in the patient's home or in hospitals where a Hawley or Albee table is not available.

It weighs only 20 lbs. and can easily be carried in one hand. It consists of a central piece which is clamped on the operating table and two steel pipes extending out to hold the foot-pieces. The foot-piece is similar to that of the Hawley table, but much lighter. The central piece is made mostly of gas pipe and is shaped approximately like a half circle which lies across the table and is held by two adjustable clamps reaching down under the edge of the table top. There are two upright non-detachable perineal bars set 1 in. apart. Clamps on the perimeter of the central piece hold the extension rods in abduction The foot-piece is held on the extension rod by a friction clutch, and extension is increased by a thumb screw.

SURGERY OF THE BLOOD AND LYMPH SYSTEMS

BLOOD VESSELS

Fry, H. J. B., and Shattock, C. E.: Sarcomatous Permeation of the Inferior Vena Cava and the Right Side of the Heart. *Brit. J. Surg.*, 1926, xiv, 337.

The authors report in detail a case of intravascular sarcomatous growth of extraordinary formation.

The patient was a primipara aged 19 years who had been recently delivered normally of a full-term child. Ten days after labor she complained of patches of numbness. There was numbness in the right leg from the hip to the knee, but none at all on the left side. Later, severe pains developed in the back and the temperature became irregular. Four months after the delivery of the child, pelvic examination revealed a large hard mass projecting forward from the sacrum, and the X-ray showed a large irregular shadow of the density of bone which was superimposed on the sacral promontory. Laparotomy revealed a large hard tumor growing from the sacrum. Section of a piece showed it to be an ossifying chondroma without malignant changes.

After the operation the patient's general condition gradually became less favorable and an enormous œdema of both lower limbs and vulva developed. Death with uræmic symptoms resulted twelve months after the birth of the child.

Autopsy revealed, arising from the sacrum, a smooth, firm growth with a slightly nodular surface, the limits of which could not be defined. The inferior vena cava was greatly distended, being a rounded semisolid pillar extending from the juncture of the common iliac veins to the lower surface of the diaphragm. It appeared to be completely thrombosed. The common iliac veins on both sides were involved and compressed by the growth. The lumen of the inferior vena cava was occupied by a firm, whitish, gristly mass which extended through the right auricle into the right ventricle and passed out as a solid cord through the pulmonary valve into the pulmonary artery. Death was due apparently more to uræmia than to the growth or its immediate effects.

Microscopic section of the growth in the inferior vena cava showed a layer of well-formed cartilage cells with minute traces of calcareous deposit at certain points and small isolated nests of sarcomatous cells.

The growth present in the vascular system as a solid mass was not less than 53 cm. long and its average diameter was 5.5 cm.

The enlargement of the veins of the anterior abdominal wall which Weber noted in thrombosis of the inferior vena cava was not a feature in this case.

JACOB S. GROVE, M.D.

BLOOD; TRANSFUSION

Mayo, W. J.: Dyscrasias of the Blood. *New Orleans M. & S. J.*, 1926, lxxix, 299.

The author discusses briefly certain fundamental changes in the blood plasma due to the excessive accumulation of catabolic substances, the result of normal as well as abnormal metabolism, which must be eliminated from the body. These disturbances are associated with alteration of the normal degree of alkalinity of the fluids of the body. The necessity for sodium chloride and a trace of iodine in the body has been handed down from our marine ancestors.

The normal balance of the constituents of the blood is in part maintained by excretions, among which are the urinary, intestinal, and biliary excretions. Interference with any of these excretions upsets the alkali-acid balance and produces the condition of alkalosis or acidosis associated with the retention of urea and creatinin and a disturbance of the concentration of chloride in the blood.

The function of the kidney is briefly described, and reference made to Cushny's hypothesis. All forms of chronic nephritis and nephrosis are not necessarily dependent on infection. If urinary drainage is interfered with, as by pressure or obstruction, the stage is set for more serious disturbances in the constitution of the blood which will quickly result in death if the condition is not relieved. The blood urea and creatinin increase and the heart becomes weak. If large quantities of a normal solution of sodium chloride and an easily digestible carbohydrate diet are given and if, in addition, a 10 per cent glucose solution is injected intravenously, as suggested by Matas, the patient will be so far restored to health that operation for correction of the obstruction may be undertaken. By these methods the mortality following operations for enlargement of the prostate has been remarkably reduced.

In the condition known as "high intestinal obstruction," whatever its cause, there is also a disturbance of the normal alkalinity of the blood and the production of alkalosis. Urea is retained and its concentration in the blood rises as does that of creatinin. Marked dehydration is due largely to the vomiting. The blood chlorides decrease as much as 50 per cent. When the alkalosis is marked, tetany may result.

Jejunostomy is a fairly reliable remedy, but the chemical disturbance can be corrected in a surprisingly short time by the intravenous introduction of solutions of sodium chloride and glucose. Reference is made to the work of Walters in demonstrating that neither chlorides nor water alone will produce this result, they must be combined in propor-

tions approximating the physiological concentration. As a result of these methods, operations on the stomach and duodenum have been performed with a notable decrease in the mortality.

When bile circulates in the blood as in jaundice, the bile acids and pigments combine with the plasma. In certain patients there may be purpuric manifestations and acidosis. With fixing of the calcium there is interference with the normal coagulability of the blood. This conception has led to the administration of calcium chloride before operations in the presence of jaundice. A carbohydrate diet with large quantities of fluid is given, sometimes combined with the intravenous administration of glucose. In the presence of acidosis, sodium bicarbonate rather than calcium chloride should be used. In many cases transfusions of blood are necessary.

Speese, J.: The Surgical Aspect of Blood Dyscrasias. *Ann. Surg.*, 1926, lxxxiv, 477.

Speese discusses certain forms of splenomegaly which are accompanied by disease of the blood and in which the attempt is made to cure or arrest the condition by removal of the spleen. The various functions of the spleen are cited. While the effect of the removal of the spleen has been studied extensively in animals and in cases of splenic rupture, Speese emphasizes that these observations have been made on healthy individuals and that when the spleen is removed for definite pathological processes the findings are very different.

With regard to pernicious anæmia attention is called to the insidious onset of the condition with pallor, achlorhydria, and gradual loss of strength. Glossitis is an early symptom. Speese is of the opinion that infection is a factor in the etiology. The improvement following splenectomy has been far greater than that produced by transfusion or other palliative methods.

Hæmolytic jaundice is characterized by splenic enlargement, an icteric tinge of the skin and scleræ, absence of bile in the urine, and the presence of bile in the stools. One of the most important findings is increased fragility of the red blood cells. The results of splenectomy in these cases are most gratifying.

Banti's disease is characterized by a clinical course in which there is a progressive increase in the severity of the symptoms. One of the first signs is enlargement of the spleen. The anæmia is of the secondary type. It is very important to operate in the early stages of the disease, before dense adhesions are formed between the spleen and the surrounding parts, particularly the diaphragm, and before the development of anæmia, liver fibrosis, and ascites.

In Gaucher's disease, splenectomy is the only method that has met with success, but it cannot be stated positively to produce a cure.

Purpura hæmorrhagica is most commonly attributed to infection. A careful blood examination is of the utmost importance. In the diagnosis, the bleeding time, coagulation time, and capillary resistance are of importance. A blood phenomenon in this condition is failure of the clots to retract. Mention is made of the acute fulminating type and the type becoming chronic which is seen most frequently in early life, particularly in girls. Little can be expected from non-operative treatment. The best chance of cure is offered by splenectomy.

In the light of our present knowledge, splenectomy is contra-indicated in lymphoid leukæmia, polycythæmia, and the rapidly progressing fulminating forms of hæmolytic jaundice, splenic anæmia, and pernicious anæmia. In myelogenous leukæmia, splenectomy offers the best chance of cure if the spleen is previously treated with radium.

EMIL C. ROBITSHEK, M.D.

Rolleston, Sir H.: Indications for Blood Transfusion. *Brit. M. J.*, 1926, ii, 969.

Cruchet, R.: Transfusion of Blood from Animal to Man. *Brit. M. J.*, 1926, ii, 975.

Keynes, G.: Blood Transfusion in Surgery. *Brit. M. J.*, 1926, ii, 980.

ROLLESTON states that the survival of transfused red blood cells in the circulation varies with the morbid condition. Reactions are often dependent upon factors other than the blood grouping. There are three forms of reactions: (1) the acute, due to agglutination and hæmolysis, which occurs immediately; (2) the delayed or proteolytic response coming on after from one to twelve hours; and (3) the systemic or constitutional reaction.

Blood transfusions are of value to make good the deficiency of blood after acute hæmorrhage, to increase the coagulability of the blood by supplying fibrinogen, to make good a deficiency of red blood cells, to furnish substances in which the blood is deficient, such as antibodies in acute infections and functionally active hæmoglobin in carbon monoxide poisoning, to act on the bone marrow which is disordered as in leukæmia, to dilute toxins in toxæmia, and to increase the bactericidal power of the blood through the action of leucocytes and the opsonic power of the serum.

In reviewing the indications for blood transfusion, Rolleston discusses acute anæmia due to hæmorrhage, chronic anæmia due to repeated hæmorrhages, hæmorrhagic diseases and conditions, purpura hæmorrhagica, postoperative shock, hæmorrhagic diseases of the newborn, hæmophilia, jaundice, pernicious anæmia, anæmias other than Addison's anæmia, leukæmia, acute septicæmia, intoxications such as carbon monoxide poisoning, and debility.

CRUCHET presents a review of experiments in the transfusion of blood from animals to man. He draws the following conclusions:

1. It is necessary to transfuse the blood very slowly.

2. The donor animal must be absolutely healthy and must not have performed any muscular work for one or two hours previously.

3. The blood must be used immediately after it has passed from the vein of the yielding animal.

4. In the transfusion of blood from animal to man the blood of the horse seems to be better tolerated than that of the sheep or the ox.

5. The blood of the horse should be diluted one-half to one-third with physiological serum.

6. It is well to add adrenalin to the mixture to be transfused up to a strength of 1:1,000.

7. The transfusion of the blood of the horse to man might be done in the same way as the intravenous injection of physiological serum.

KEYNES summarizes the reports received by the London Blood Transfusion Service. Two hundred and six of the cases in which transfusion was done were medical and 411 were surgical.

Gastric and duodenal conditions, 121 cases: no benefit in eighteen; temporary benefit but subsequent death in thirteen; a good or very good result in ninety.

General surgical cases, including amputations, splenectomy, etc., 100 cases: no benefit in four; temporary improvement in six; a good or very good result in ninety.

Hæmophilia, four cases: no benefit in one; a good result in three.

Septicæmia and pyæmia, twenty-five cases: no benefit in twenty-two; a good result in three.

Gynecological conditions, sixty-nine cases: no benefit in seven; temporary improvement in four; a good or very good result in fifty-eight.

J. FRANK DOUGHTY, M.D.

Grulee, C. G.: Intraperitoneal Transfusion in von Jaksch's Anæmia. *N. York State J. M.*, 1926, xxvi, 921.

Von Jaksch's anæmia, or anæmia pseudo-leukæmia infantum, is a severe anæmia accompanied by splenic enlargement, an increase in the white blood cells, chiefly the mononuclears, enlargement of the liver, and nucleated red cells.

The author reports upon a series of twelve cases in infants from 9 months to 3 years of age which were treated by intraperitoneal transfusion. Both the direct and the indirect method were used.

In the direct method, the blood was drawn from the donor into a 100-c.cm. glass syringe and injected into the peritoneal cavity immediately. When more than 100 c.cm. was given, two syringes were employed.

In the indirect method the blood was drawn from the donor into a bleeding bottle and mixed with 10 c.cm. of a 2½ per cent solution of sodium citrate for each 100 c.cm. of blood.

The immediate results were good in almost all of the cases, improvement being noted in both the blood picture and the general condition. The ultimate results were not as definite. The younger the child the better the result.

Intraperitoneal transfusion permits the introduction of large quantities of blood at short intervals without much difficulty.

RAYMOND GREEN, M.D.

SURGICAL TECHNIQUE

OPERATIVE SURGERY AND TECHNIQUE; POSTOPERATIVE TREATMENT

Rehn, E.: Surgery and Organ Function (Chirurgie und Organfunktion). *50 Tag. d. deutsch. Ges. f. Chir.*, Berlin, 1926.

Organ function is life. Life depends not only on the function of the organs, but also on their harmonious function. Disease represents the sum of functional disturbances becoming progressively more extensive. The surgeon must attempt to give his treatment before the development of secondary organic disturbances. Therefore an early diagnosis is of great importance. In the majority of cases, only an early operation will effect a cure.

The surgeon must bear in mind that an operation is of more importance to the patient than the results of the mechanical injury. The regeneration following an operation is not limited to the site of the operation, but occurs in all of the organs. Every physical change is accompanied by a psychic change. The surgeon must not be a mere technician; he must always take into consideration the effect of the operation on the organism as a whole. Different persons react differently to operation. The surgeon must know the various types of reaction and attempt to prevent their harmful effects.

The importance of the constitution in the effect of an operation was especially emphasized by Payr. In the cases of hypoplastic and lymphatic persons the reaction is such that every operation is dangerous. Asthenic and hyposthenic persons are less endangered. Another type endangered by operative interference is the embolic type. The embolic type of person has a pale skin and adiposity and is not well adapted to withstand mental and physical strain. By means of comparative graphs, Rehn demonstrates the increased coagulability of the blood of such persons. This type of patient should maintain physical activity as much as possible up to the time of operation.

Endocrine disturbances, constitutional anomalies of the sympathetic nervous system with their effect on the respiration, heart, blood pressure, and metabolism are of influence on the operative course. The sympathetic as well as the parasympathetic nervous system exerts an influence. Weakening of one of these systems is associated with danger.

Complete confidence of the patient in the physician aids convalescence. The vagotonic person is a poor operative risk. Such a person has an increased susceptibility to shock due to lability of the vasomotor system. Under such circumstances a fall in the blood pressure occurs easily and even local anæsthesia is to be avoided. The vagotonic type has slight resistance to the injurious effects of narcosis; there occurs a disturbance in the relations between the potassium and the calcium salts to the detriment of the latter. The unfavorable effects of this disturbance of balance may be kept within certain bounds by means of afenil.

Susceptibility to shock is increased also in the young child. Shock may be caused by any disturbance of the mineral metabolism. Its occurrence is favored also by all substances of a toxic or infectious nature. The danger of narcosis is not to be underestimated. Narcosis may be the cause of a vasomotor collapse which has a very unfavorable effect on the heart and the circulation. It leads also to severe disturbances of metabolism which are manifested chiefly in the liver and biliary passages. Therefore chronic disease of the liver is especially aggravated by narcosis.

Investigations have shown that the danger of narcosis is caused chiefly by a disturbance of the alkali reserve. Experiments on animals show that in narcosis induced with chloroform or ether there is a decrease in the alkali reserve. Most favorable in this respect is narcylen. Attempts should be made to render this substance less explosive.

In the treatment of shock and the other complications of narcosis it is wrong to use saline or glucose infusions indiscriminately. In many cases a transfusion of blood is preferable.

Before every operation a functional test of the heart, lungs, kidneys, and liver should be made when possible. The author has made graphic studies by means of the electrocardiogram before, during, and after operation. Disturbances of cardiac conduction, extrasystoles, and auricular fibrillation may develop. The effect depends upon the condition of the heart, especially the functional capacity of the heart muscle. Therefore a functional study of the heart is of importance. A good test is that recommended by Kaufmann at the Surgical Congress in Germany last year, combined with the blowing up of a tube with air ten times. To stimulate the function of the heart, digitalis is recommended, but should not be given indiscriminately in every case.

The chief danger in the lungs is the development of capillary bronchitis, in the origin of which the irritation of the sympathetic nervous system plays an important part. As prophylactic treatment, the administration of afenil is recommended. Although this does not always prevent pulmonary complications, it renders them less severe.

The kidneys always show an acetonuria and at times a transitory albuminuria after narcosis. For the testing of kidney function the author recommends his alkali-acid test.

The results of the author's tests of liver function show how serious the disturbances following nar-

cosis may be, especially in cases of chronic liver disease. In the latter, great care is necessary.

All of the facts cited demonstrate that the surgeon must have an accurate knowledge of physiology. Each organ is a part of the whole. In the development of the surgery of the individual organs the body as a whole must not be forgotten. The more important the diseased organ is to the body, the more conservative must be the surgical treatment.

In the healing wounds the total metabolism and the distribution of electrolytes play an important rôle. Cell life and cell function lead to healing, but are themselves dependent upon higher factors. In the treatment of wounds, local applications are often of less importance than regulation of the general habits of life. The author cites the research of the Munich school with regard to the effect of foods. In these investigations it was found that wounds heal better when acid foods are given than when alkaline foods are given. In cachexia, the conditions are different, the patient suffering from a depletion of the alkali reserve. In colon bacillus pyelitis and many conditions associated with the formation of urinary sand, an intake of alkali must be provided. In the case of a poorly healing wound in a diabetic, local treatment will not help if the sugar metabolism is not regulated by the administration of insulin. The newborn infant has a tendency toward acidosis. This must be combated.

In conclusion Rehn states that surgery will progress only on the basis of a physiological study of the interrelations of the parts of the body.

STETTINER (Z).

PHYSICOCHEMICAL METHODS IN SURGERY

ROENTGENOLOGY

Putnam, T. J.: Some Brominized Oils for Radiographic Use: Preliminary Report. *J. Am. M. Ass.*, 1926, lxxxvii, 1102.

The various radio-opaque oils (lipiodol, iodipin, etc.) which have recently been introduced find a growing use in the roentgenography of the spinal canal, the ventricles, the lungs, the paranasal sinuses, the joints, the uterus, and the tubes. But though various accidents have been reported from their use, no detailed study of their composition and action has been made. Both of the original oils are iodized salad (vegetable) oils, introduced as palatable iodides many years ago, and their introduction into roentgenography has been largely empirical.

Several different types of bromine and iodine compounds were made from eleven kinds of oil selected for their variety of physical and chemical properties.

The irritating qualities of the more promising compounds were tested by injecting 0.05 c.cm. of each into the anterior chamber of a cat's eye. After twenty-four hours, the aqueous humor was withdrawn under standard conditions and the cells in it were counted. It was found that the commercial iodized vegetable oils gave counts of 2,000 to 5,000. The opaque oil which appeared the least irritating was brominized lard oil, which produced counts of 150 to 500 cells under similar circumstances. Small bubbles of air under the same condition gave counts of 2,000 to 8,000.

In regard to absorbability, the brominized lard oil also appeared preferable to the vegetable oils. When injected into the eye of a rat, a drop of brominized lard oil was absorbed in about four weeks while similar drops of the vegetable oils appeared intact three months later. Gases have the advantage of being more rapidly absorbed than oil.

The brominized lard oil is therefore being subjected to clinical tests. It is slightly less opaque than the oils now on the market, but is more fluid and more easily emulsified. Brominized sperm oil is very opaque and has a low viscosity, but is more irritating than lard oil.

Desjardins, A. U.: Stimulation and Immunity in Radiotherapy. *J. Am. M. Ass.*, 1926, lxxxvii, 1537.

The idea has become widespread among physicians that radium and roentgen rays possess the power of stimulating living cells, meaning thereby the growth of cells or collections of cells, such as tumors. From a survey of the literature and the amount of evidence indicating that continued acceleration of cellular metabolism does not follow irradiation, the author draws these conclusions:

1. Cells and collections of cells, such as organs or tumors, in plants and animals, cannot be stimulated in the sense that they acquire greater ability to function in a normal manner.

2. Any such apparent stimulation is only a transient phase which is invariably followed by more or less pronounced functional or organic deterioration.

3. The assertions of a few authors that such stimulation takes place must be based on the insufficient duration of their experiments, the misinterpretation of the results of their studies, complicating circumstances, the nature of which is not clear, or misuse of the word "stimulation."

Another misconception which has gained wide acceptance by members of the medical profession concerns the biological effects produced by irradiation or, more specifically, the mechanism by which radium and roentgen rays bring about the partial regression or the complete disappearance of tumors. The mass of evidence shows that the major factor in the effect of radium and roentgen rays on cancer cells is a direct one. It consists of at least two overlapping and interrelated factors: (1) a direct action on the cells of the tumor, and (2) a direct action on the endothelial cells in the blood vessels supplying the tumor. Such of these cells as are irreparably injured by the irradiation are replaced by connective tissue. In some tumors the action of the rays is greatest on the tumor cells themselves, whereas in others the influence on the vascular endothelium predominates. To these major factors there may be added still others, though less important, but in the present state of our knowledge it is impossible to specify what these may be and what limited influence they may have in the action of irradiation on living tissues.

MISCELLANEOUS

Mertz, A.: An Increase of Ferment in the Serum Following Light and Roentgen-Ray Irradiation, an Index of the Processes of Cell Destruction (Fermentvermehrung im Serum nach Licht- und Roentgenbestrahlungen, ein Index fuer Zellzerfallsvorgaenge). *Strahlentherapie*, 1926, xxii, 301.

Peptidase, a ferment liberated by the breaking down of the dipeptid glycyltryptophan, occurs in the living body cells and after the physiological breaking down of the cells goes over into the blood serum. It can be easily demonstrated in the serum by incubating the reagent in the incubator at 56 degrees for two and a half hours. The tryptophan thus freed can be distinguished by means of the red color which appears when it is treated with chlorine or bromine. Graded dilutions of the serum are made and the solution of the greatest dilution which still shows a

breaking down is recorded. In this way a peptolytic index is obtained which in each individual is strikingly constant.

In the cases of children in which this index was determined before and after irradiation with the Alpine sun rays, natural sunlight, and roentgen rays, a distinct increase in the index was noted after the irradiation. The author believes that illness following roentgen-ray irradiation may be regarded as a poisoning by protein end-products.

VON SCHUBERT (G).

Hess, A. F.: The Use and Misuse of Ultraviolet Therapy. *N. York State J. M.*, 1926, xxvi, 816.

Ultraviolet irradiation is a specific for the prevention and cure of rickets and tetany. By exposure to ultraviolet irradiation, it is possible to convert foods such as vegetable oils, carrots, spinach, and dried milk into active anti-rachitic substances. Ultraviolet irradiation is of great value also in the treatment of multiple furunculosis and tuberculosis, especially tuberculosis of the peritoneum, bones, joints, and glands. Brunettes seem to be more benefited by it than blonds. In eczema the results of this treatment have been unsatisfactory.

LLEWELLYN R. LEWIS, M.D.

Païzis, D.: The Treatment of Moist Gangrene in Diabetics by Diathermy (Traitement des gangrènes humides des diabétiques par la diathermie). *Presse méd.*, Par., 1926, xxxiv, 1242.

Diathermy is effective in moist gangrene in diabetics because it produces heat in the tissues, improves the circulation, increases the defensive power of the body against pathogenic agents either by improving the local circulation or acting directly on the cells, and may act also directly on the bacteria. Of nine cases treated by the author with this method, a completely successful result was obtained in eight. From the very beginning of the treatment Païzis noted a decrease in the pain and œdema, the elimination of sloughs, intense granulation of the wounds, a decrease in the general signs of toxi-infection, and a fall in the temperature. This improvement did not seem to depend on the insulin which some of the patients had been given.

When the gangrene is situated on the foot an electrode of very thin paper such as that used to wrap chocolates is wrapped around the upper part of the leg and the front part of the foot is sheathed to form another electrode. Care is taken to apply the paper tightly to the skin for if it is separated at any point a spark will be produced which will cause a troublesome burn. As it is sometimes difficult to apply the electrodes closely, Chevalier tried to overcome this difficulty by using as an electrode a saturated solution of sodium chloride into which he plunged the patient's foot. The current varies 500 to 1,200 ma., depending on the patient's tolerance, and is applied at first for twenty minutes twice a day. The intervals are then increased gradually to possibly two or three days.

The strictest asepsis is observed in the dressings. Wet dressings are used in the beginning, and dry dressings are applied when the inflammation has subsided. Insulin and a diabetic diet should be given according to the usual rules.

AUDREY G. MORGAN, M.D.

MISCELLANEOUS

GENERAL BACTERIAL, PROTOZOAN, AND PARASITIC INFECTIONS

Kaiserling: Sepsis from the Pathologico-Anatomical Standpoint (Sepsis vom pathologisch-anatomischen Standpunkt). *Deutsche med. Wchnschr.*, 1926, lii, 1199.

By "sepsis" the author understands the reaction of the body to the continued or repeated introduction into the blood stream of preponderantly pus-forming micro-organisms. The reaction is dependent upon the virulence and the number of the organisms and the ability of the body to react.

Sepsis maligna occurs when virulent organisms enter a body incapable of resistance. As death results after one or two days, the changes in the internal organs are of no significance.

Gas-forming micro-organisms, on the other hand, cause very marked changes. Worthy of note in such cases is a peculiar warmth of the corpse. The growth of the organisms progresses rapidly in the corpse; therefore sections yield little worthwhile information when they are made later than one hour after death. The spleen is septic. The liver shows the picture of cloudy swelling, and occasionally there is a light icterus. The icterus is not always due to changes in the liver; on the contrary, it is often the result of increased hæmolysis in the blood, spleen, and liver. The kidneys and heart muscle show cloudy swelling. On microscopic examination an increase in the leucocytes in the blood is found. A cure occurs if the body is able to wall off the portal of entry by new connective tissue and to kill the bacteria that have already entered it.

Streptococci often cause less cell and tissue damage than staphylococci and produce a diffuse phlegmon instead of numerous abscesses. Endocarditis and thrombophlebitis are more common in staphylococcal infections and are dangerous especially because of the necrosis and extensive breaking down of the tissues; they often lead to embolism.

It appears that the chief rôle in the defense against the micro-organisms is taken by the vascular endothelium in the liver, spleen, and bone marrow and the reticulo-endothelium and the endothelium of the vessel walls which are morphologically and functionally related to it.

Of the septic types of endocarditis, verrucous endocarditis is the least dangerous. To this belongs the disease of the valves developing especially in the course of rheumatic arthritis. In such cases of chronic sepsis we are dealing with a smaller number of bacteria which, because of the good resistance offered by the endothelium and the connective tissue, produce only limited necrosis and small blood-platelet thromboses, and by irritation stimulate the growth of connective tissue. Related to endocarditis are the rare cases of endarteritis ulcerosa.

FREY (Z).

Nickel, A. C.: The Localization in Animals of Bacteria Isolated from Foci of Infection. *J. Am. M. Ass.*, 1926, lxxxvii, 1117.

The foci of infection studied by the author were the tonsils, teeth, prostate, and cervix. All culture material obtained from these foci was plated aerobically on blood-agar plates and inoculated into glucose-brain agar and glucose-brain broth made according to Rosenow's method. The great majority of the strains isolated consisted of a partial tension, green-producing streptococcus which in primary culture grew poorly or not at all under aerobic conditions. These partial tension cultures, when freshly isolated and injected intravenously into rabbits, tended to produce lesions similar to the lesions of the patient from whom the culture was obtained.

The highest incidence of specific localization was shown by cultures from infected teeth. In every instance the percentage of localization in the control animals was only a small fraction of the large percentage of specific localization in the animals with the specific strains. The causal relationship between the organisms and the lesions produced was established by isolation of the former from the lesions when the blood and other tissues proved sterile and by their demonstration in the sections. The diseases thus studied included arthritis, myocarditis and endocarditis, myositis, lesions of the eye, lesions of the skin, and ulcer of the stomach and duodenum.

This method of estimating the localizing power in animals of organisms isolated from foci of infection is of diagnostic as well as therapeutic value. It often serves to demonstrate which of a series of organisms commonly found in foci bears a causal relationship to the systemic disease and what particular focus harbors such organisms, and it provides the means for active immunization with specific autogenous vaccines.

Jackson, R. H.: Surgical Treatment of Certain Massive Blastomycotic Skin Lesions. *Am. J. Surg.*, 1926, n.s. i, 185.

Jackson reports two cases of extensive blastomycotic skin lesions in which surgical intervention was undertaken after the patients had been under the care of competent dermatologists for eleven and two years respectively and the lesions had become more extensive under non-surgical treatment.

Under nitrous oxide and ether anæsthesia, large soldering irons heated to a dull red were applied

and re-applied to the entire area of the lesion until a thorough cooking of the tissues to a depth of about 3 or 4 cm was assured. The cooked surface was then removed with sharp spoon curettes. The curetting continued until blood appeared, when the irons were again applied. This procedure was repeated until all evidence of the disease had been removed. The final application of the cautery left a light charred, seared area.

At the conclusion of the operation a hot wet dressing of 1 per cent copper sulphate solution was applied to the whole area. This dressing was renewed each day until healthy granulations and epithelization appeared. Potassium iodide, 1 dr., and ¼-gr. capsules of copper sulphate (precipitated) were administered internally.

Epithelization was aided by the application of adhesive zinc oxide plaster strips. Skin grafting was not necessary. In one case epithelization was nearly complete at the end of seven weeks. In the other, convalescence was shortened by the use of "cootie" skin grafts.

With regard to the diagnosis of blastomycosis, Jackson mentions the peculiar foul odor occurring in advanced cases. JACOB M. MORA, M.D

DUCTLESS GLANDS

Rowntree, L. G., and Snell, A. M.: The Diagnosis and Treatment of Certain Glandular Deficiencies. *Med. Clin. N. Am.*, 1926, x, 513.

The treatment of endocrine diseases at the present time resolves itself into: (1) substitution therapy, that is, the replacement of a partially or completely missing hormone by one of the same nature, or (2) the surgical removal of gland tissue which is excreting an excess of hormone. A perfect example of substitution therapy is the management of a case of myxœdema. In this condition sufficient thyroid extract is given to bring the depressed metabolic rate to normal and a suitable maintenance dose of thyroid extract is given daily in order to keep it at a normal level.

Parathyroid deficiencies, either acute or chronic, are frequently due to the accidental surgical removal or injury of the parathyroid bodies. The patient responds favorably to substitution therapy in which the parathyroid extract of Collip is used. Two cases are presented, one of acute tetania parathyreopriva and the other of chronic tetany. In both the symptoms were relieved by the use of parahormone together with large doses of calcium lactate, a normal calcium equilibrium was established after a considerable period, and it was possible to discontinue the treatment. In experimental animals, a similar phenomenon is observed.

Diabetes insipidus may be either idiopathic or secondary to cerebral lesions of various sorts. In the latter group metabolic rates are, as a rule, lowered, while in idiopathic cases the rates are usually within normal limits. This point is frequently of diagnostic value. Secondary diabetes insipidus due to syphilis of the central nervous system is rarely relieved by vigorous antisyphilis treatment. The use of pituitary extract is effective in both types of the disease and controls the thirst and polyuria although it accomplishes nothing in a curative way. It is important to try various commercial preparations of pituitrin both hypodermically and intranasally since there is a marked individual variation in the response to various extracts.

Addison's disease is probably due to a deficiency of the cortical portion of the suprarenal rather than to a medullary lesion. Preparations of the cortex in use at the present time do not seem to be active, or at least do not appreciably prolong the life of animals from which the suprarenals have been removed. Substitution therapy in Addison's disease is therefore confined to the administration of epinephrin by hypodermic injection and by rectum to the point of tolerance. The administration of suprarenal cortex by mouth should be tried, even though there is some uncertainty as to its effect. The treatment of collapse, so frequently a terminal event in this disease, is rarely successful. Protection of the patient against exposure, infection, and emotional disturbances which may precipitate such incidents is of great importance. It is difficult to evaluate the effect of organotherapy in Addison's disease. Such treatment appears to do much good in certain cases but in others has little effect. Of a group of sixty-eight patients, three lived nearly four years, and in a number of those more recently treated the results of such treatment are apparently stationary.

BIBLIOGRAPHY *of* CURRENT LITERATURE

NOTE.—THE BOLD FACE FIGURES IN BRACKETS AT THE RIGHT OF A REFERENCE INDICATE THE PAGE OF THIS ISSUE ON WHICH AN ABSTRACT OF THE ARTICLE REFERRED TO MAY BE FOUND.

SURGERY OF THE HEAD AND NECK

Head

The chiasmatic syndrome in fractures of the skull. J. OKINCZYC and G. RENARD. Bull. et mém. Soc. nat. de chir., 1926, lii, 905.

A case of lateral sinus thrombosis with organized clot: simple exposure; recovery. H. W. A. POST. Guy's Hosp. Rep., Lond., 1926, lxxvi, 481.

The pathological examination in cavernous sinus thrombosis as a guide to the diagnosis, prognosis, and treatment. O. J. DIXON. J. Am. M. Ass., 1926, lxxxvii, 1088. **[259]**

Pre-auricular fistulæ. F. A. R. STAMMERS. Brit. J. Surg., 1926, xiv, 359. **[259]**

Observations on salivary secretion and associated clinical findings. R. A. BARLOW. Ann. Otol., Rhinol. & Laryngol., 1926, xxxv, 1021.

Tumors of the parotid region. BRODIER. Bull. et mém. Soc. d. chirurgiens de Par., 1926, xviii, 585.

Mixed tumors of the parotid gland and their removal, with a consideration of the seventh nerve. L. W. GROVE. South. M. J., 1926, xix, 752. **[259]**

Genital cancer and lymphogranulomatosis of the parotid gland. W. FISCHER. Deutsche Ztschr. f. Chir., 1926, cxcvii, 242.

Skeletal adjustment in jaw growth. T. W. TODD. Dental Cosmos, 1926, lxviii, 1168.

Two cases of fracture of the lower jaw in children. H. BOUVIER. Ztschr. f. Stomatol., 1926, xxiv, 526.

A new method of anæsthesia of the region of the upper and lower jaw. A. LINDEMANN. Deutsche Monatsschr. f. Zahnh., 1926, xliv, 387.

The exposure of the temporomandibular joint. K. MERMINGAS. Zentralbl. f. Chir., 1926, liii, 1510.

Actinomycotic infection of the jaw: a clinical and pathological study. G. A. CHIURCO. Ztschr. f. Stomatol., 1926, xxiv, 763.

Ankylosis of the jaw. PATEL. Lyon chir., 1926, xxiii, 611.

Two unusual tumors of the lower jaw. SONNTAG. Zentralbl. f. Chir., 1926, liii, 2543.

Carcinoma of the lower jaw. D. QUICK. Am. J. Surg., 1926, n.s. i, 360.

Eye

Industrial ophthalmology. C. A. BAHN. New Orleans M. & S. J., 1926, lxxix, 429.

Tolerance to foreign bodies within the posterior segment of the eye. A. E. BULSON, JR. J. Am. M. Ass., 1926, lxxxvii, 2071.

Electromagnet extraction of foreign bodies from the eye. A. W. MORSE. Northwest Med., 1926, xxv, 667.

Routine eye examination. I. C. MANN. Brit. M. J., 1926, ii, 1055.

Unlearnable prism test for suspected malingering. E. H. SCHILD. Am. J. Ophth., 1926, 3 s. ix, 741. **[260]**

Diagnostic methods, perimetry neglected. W. F. SWETT. Am. J. Ophth., 1926, 3 s. ix, 903.

Stereoscopic vision. E. D. CANEJA. Med. Ibera, 1926, x, 283.

Slit-lamp technique applied to simple apparatus. T. H. BUTLER. Brit. M. J., 1926, ii, 1104.

Refraction under cyclodamia. D. SMITH. Am. J. Ophth., 1926, 3 s. ix, 896.

The effect of prolonged application of cold on the eye. K. E. MANDAN. Indian M. Gaz., 1926, lxi, 600.

Headaches, with especial reference to those of ocular origin. G. E. DE SCHWEINITZ. Canadian M. Ass. J., 1926, xvi, 1314.

Traumatic enophthalmos. F. A. MORRISON and C. W. RUTHERFORD. Am. J. Ophth., 1926, 3 s. ix, 876.

Institutional treatment of eye disease in children. Brit. J. Ophth., 1926, x, 651.

Higher education of girls with little or no sight: Chorley Wood College. N. B. HARMAN. Brit. M. J., 1926, ii, 1103.

Intra-ocular sarcoma in children: a clinical and pathological study of ten cases. W. R. PARKER and W. H. STOKES. J. Am. M. Ass., 1926, lxxxvii, 1891.

The mucous membrane from the middle turbinate in plastic surgery of the eye. M. WIENER. J. Am. M. Ass., 1926, lxxxvii, 2091.

The travelling trachoma clinic. C. B. KOBERT. Kentucky M. J., 1926, xxiv, 616.

Granular conjunctivitis in the rabbit and the origin of trachoma. C. NICOLLE. Bol. inst. de clin. quir., 1926, ii, 243.

The rôle played by epithelium in infections of the conjunctiva and cornea. W. C. FINNOFF. Colorado Med., 1926, xxiii, 387.

Congenital muscular squint. L. G. DODDS. Brit. J. Ophth., 1926, x, 649.

The treatment of lachrymal obstruction. A. E. ILES. Bristol M.-Chir. J., 1926, xliii, 191.

Dacryocystorrhinostomy. J. BASTERRA. Med. Ibera, 1926, x, 249.

Dacryocystorrhinostomy. J. B. SANTACRUZ. Noticias med., 1926, i, 39.

Further studies on the light sense in early glaucoma. G. S. DERBY, J. H. WAITE, and E. B. KIRK. Arch. Ophth., 1926, lv, 575.

Atypical glaucoma. J. B. STANFORD. New Orleans M. & S. J., 1926, lxxix, 425.

Central choroiditis. R. H. T. MANN. Texas State J. M., 1926, xxii, 518.

Hæmangioma of the choroid. E. J. LENT and M. B. LYON. J. Indiana State M. Ass., 1926, xix, 443.

A case of essential megalocornea. CASTROVIEJO. Med. Ibera, 1926, x, 309.

Steamy cornea in cases of non-inflammatory increased tension. G. YOUNG. Brit. M. J., 1926, ii, 1106.

The probable etiological relationship between keratitis and myopia. L. A. CHÁVEZ VELANDO. Crón. méd., Lima, 1926, xliii, 265.

Recurrent keratitis profunda from maxillary sinusitis. A. W. WILDE. Am. J. Ophth., 1926, 3 s. ix, 892.

Band keratitis. A. J. BEDELL. Am. J. Ophth., 1926, 3 s. ix, 887.

The aqueous humor. A. M. YUDKIN. J. Am. M. Ass., 1926, lxxxvii, 1910.

Recurrence of iritis as influenced by the removal of infections: summary of fifty cases. E. E. IRONS and E. V. L. BROWN. J. Am. M. Ass., 1926, lxxxvii, 1167. **[260]**

The prostate as a remote focus of infection in ocular inflammations. W. ZENTMAYER. J. Am. M. Ass., 1926, lxxxvii, 1172. **[260]**

Ocular disease occurring in the course of non-dysenteric amœbiasis. L. MILLS. J. Am. M. Ass., 1926, lxxxvii, 1176. **[260]**

Tuberculous iridocyclitis as observed with the slit lamp, with remarks on tuberculin treatment. C. KING. Arch. Ophth., 1926, lv, 563.

Tuberculous iridocyclitis as observed with the slit lamp. C. KING. Ohio State M. J., 1926, xxii, 939.

The action of pilocarpine on the rat's pupil. J. A. WADDELL. J. Lab. & Clin. Med., 1926, xii, 232.

Blocking of the main trunk of the facial nerve in cataract operations, based on experience in over 150 cases. R. E. WRIGHT. Arch. Ophth., 1926, lv, 555.

The extraction of a senile cataract in a chronic glaucomatous eye with subsequent iridotomy. P. E. SUEHS. Texas State J. M., 1926, xxii, 515.

An untied conjunctival suture and conjunctival bridge in cataract extraction: results and conclusions. C. BERENS and R. R. LOSEY. Brit. J. Ophth., 1926, x, 577.

Complications in cataract surgery—case report. W. D. ROWLAND. J. Ophth., Otol. & Laryngol., 1926, xxx, 406.

Papillœdema from the viewpoint of the otologist. E. B. DENCH. Laryngoscope, 1926, xxxvi, 857.

Papillœdema from the viewpoint of the rhinologist. E. R. FAULKNER. Laryngoscope, 1926, xxxvi, 873.

Blood pressure in the central artery of the retina. E. LIDA and E. ADROGUÉ. Semana méd., 1926, xxxiii, 1129.

The influence of negative pressure in the sphenoid on the optic nerve. L. E. WHITE. Boston M. & S. J., 1926, cxcv, 1195.

Ear

Some otolaryngological problems. R. C. LYNCH. South. M. J., 1926, xix, 893.

Fads and fancies in the practice of otolaryngology. G. E. SHAMBAUGH. J. Am. M. Ass., 1926, lxxxvii, 1720.

The relation of tactile impression and hearing perception. M. A. GOLDSTEIN. Laryngoscope, 1926, xxxvi, 729. **[261]**

Auditory sensibility. V. RIBON. Rev. med.-quirurg. hospitales, 1926, ii, 343.

Deafness from the standpoint of prevention. L. E. HETRICK. J. Ophth., Otol. & Laryngol., 1926, xxx, 413.

Diagnostic school clinic in the public schools as a factor in the conservation of hearing. H. NEWHART. J. Am. M. Ass., 1926, lxxxvii, 1882.

Three million deafened school children. E. P. FOWLER and H. FLETCHER. J. Am. M. Ass., 1926, lxxxvii, 1877.

The treatment of lateral pharyngeal bands for deafness by the X-ray-result of 100 cases. D. C. SMYTH. Ann. Otol., Rhinol. & Laryngol., 1926, xxxv, 1157.

An improved aural speculum. G. L. RICHARDS. Arch. Otolaryngol., 1926, iv, 525.

The saccus endolymphaticus and its relation to labyrinthine muscle tonus. H. G. LA REAU. Ann. Otol., Rhinol. & Laryngol., 1926, xxxv, 1032.

Ablation experiments on the labyrinth of frogs. J. TAIT. Laryngoscope, 1926, xxxvi, 713. Arch. Otolaryngol., 1926, iv, 281. **[261]**

An aqueduct in the bird's labyrinth not previously recorded, and its evolutionary significance. A. A. GRAY. J. Laryngol. & Otol., 1926, xli, 790.

The interpretation of vestibular nystagmus. P. L. ERRECART. Semana méd., 1926, xxxiii, 1067.

Neurolabyrinthitis of focal infection origin in a case with a+4 Wassermann reaction. G. W. MACKENZIE. J. Michigan State M. Soc., 1926, xxv, 619; Laryngoscope, 1926, xxxvi, 878.

Perforation of the fenestra rotunda for therapeutic purposes. C. BIEHL. J. Laryngol. & Otol., 1926, xli, 637. **[262]**

The indications for opening the labyrinth. R. LEIDLER. J. Laryngol. & Otol., 1926, xli, 641. **[262]**

The probable pathway of infection in a case of Gradenigo's syndrome. J. ADAM. J. Laryngol. & Otol., 1926, xli, 801.

The insidious, symptomless, destructive effect of cholesteatome. H. I. LILLIE and W. B. STARK. Surg. Clin. N. Am., 1926, vi, 1359. **[263]**

The home management of cases of acute otitis media. F. R. PACKARD. Therap. Gaz., 1926, 3 s. xlii, 850.

Zinc ionization in the treatment of cases of purulent otitis media. H. L. MCLAURIN. Texas State J. M., 1926, xxii, 519.

Nascent iodine in otorrhœa. F. P. STURM. Brit. M. J., 1926, ii, 1118.

Unusual symptoms and complications of mastoid disease. J. A. MORGAN. Ann. Otol., Rhinol. & Laryngol., 1926, xxxv, 1130.

Primary osteomyelitis of the mastoid bone (traumatic) with the report of a case and a review of the literature. M. E. KAHN. Ann. Otol., Rhinol. & Laryngol., 1926, xxxv, 1246.

A case of subdural abscess following an operation on the mastoid process; recovery. H. W. A. POST and W. M. MOLLISON. Guy's Hosp. Rep., Lond., 1926, lxxvi, 479.

Nose and Sinuses

Unusual congenital malformations of the nose: (1) fibrolipoma of the nasal septum; (2) bifurcated nose with a fibrolipoma of the dorsum of the nose. N. FEYGIN. Zentralbl. f. Chir., 1926, liii, 1686.

Artificial nose. C. H. CLOVIS and E. C. ARMBRECHT. Dental Cosmos, 1926, lxviii, 1198.

The sorrows of the septum. W. J. HORNE. Brit. M. J., 1926, ii, 1172.

A theory of deviated nasal septum. E. M. MIKKELSEN. Illinois M. J., 1926, l, 506.

The question of operation for nasopharyngeal fibroma. A. ZIMMERMANN. Beitr. z. Anat., Physiol., Path. u. Therap. d. Ohres etc., 1926, xxiii, 698.

The Halle operation for ozæna. R. A. BARLOW. Boston M. & S. J., 1926, cxcv, 1202.

Foreign bodies in the accessory nasal sinuses. M. B. WILSON. Ann. Otol., Rhinol. & Laryngol., 1926, xxxv, 1259.

Unrecognized nasal sinusitis. I. W. VOORHEES. Med. J. & Rec., 1926, cxxiv, 741.

Localized influenzal affections of the head. R. JACKSON. New Orleans M. & S. J., 1926, lxxix, 409.

The diagnosis of disease of the nasal accessory cavities. C. E. BOWLES. Virginia M. Month., 1926, liii, 599.

Nasal sinusitis as a cause of toxæmia. Sir W. WILLCOX. Proc. Roy. Soc. Med., Lond., 1926, xix, Sect. Laryngol., 49. **[263]**

The local administration of bacterial vaccines in the treatment of subacute and chronic nasal sinus conditions. H. Hays. Laryngoscope, 1926, xxxvi, 812.

Frontal sinus disease; three unusual consecutive cases. L. M. Hurd. Ann. Otol., Rhinol. & Laryngol., 1926, xxxv, 1093; Laryngoscope, 1926, xxxvi, 894.

Gunshot wounds of the maxillary sinus. H. F. Lampe. Ann. Otol., Rhinol. & Laryngol., 1926, xxxv, 1135.

A new procedure in the treatment of chronic maxillary sinus suppuration in children. A. M. Alden. Arch. Otolaryngol., 1926, iv, 521.

Radio-active substances; their therapeutic uses and applications; the radiation of malignancy of the maxillary sinus. J. Muir. Radiology, 1926, vii, 507.

Two cases of apparent cure by operation of a malignant growth of the antrum and ethmoid. S. L. A. Clarke and W. M. Mollison. Guy's Hosp. Rep., Lond., 1926, lxxvi, 477.

The embryology, anatomy and physiology of the ethmoid and sphenoid sinuses. F. E. Lejeune. Laryngoscope, 1926, xxxvi, 889.

The pathology of spheno-ethmoidal sinusitis. B. J. McMahon. Arch. Otolaryngol., 1926, iv, 310. [264]

Physiotherapy treatment of ethmoiditis. C. R. Brooke. Med. J. & Rec., 1926, cxxiv, 685.

External operation on the ethmo-sphenoid-frontal group of sinuses under local anæsthesia: the technique for the removal of part of the optic foramen wall for the relief of the pressure on the optic nerve. E. C. Sewall. Arch. Otolaryngol, 1926, iv, 377.

Perisinus abscess. E. P. Blashki. Med. J. Australia, 1926, ii, 803.

Tutocaine: a local anæsthetic in rhinolaryngology. O. D. Cunningham. Laryngoscope, 1926, xxxvi, 837.

Mouth

A case of plastic substitution for the upper and lower lip. R. Demel. Deutsche Ztschr. f. Chir., 1926, cxcvi, 210.

Chronic (streptococcic) hypertrophy of the lower lip. L. G. Beinhauer. Med. J. & Rec., 1926, cxxiv, 755.

On harelip and other developmental lesions of the face. W. Rankin. Glasgow M. J., 1926, n. s. xxiv, 350.

A case of hereditary harelip. G. Lévy. Bull. Soc. d'obst. et de gynéc. de Par., 1926, xv, 511.

Congenital cleft lip and palate. H. P. Ritchie. Minnesota Med., 1926, ix, 664.

Cleft palate. W. L. Shearer. Nebraska State M. J., 1926, xi, 462.

The surgical treatment of cleft palate. G. V. I. Brown. J. Am. M. Ass., 1926, lxxxvii, 1379. [264]

A septal flap in the closure of unilateral clefts of the palate. J. E. Thompson. J. Am. M. Ass., 1926, lxxxvii, 1384. [264]

A comparative study of the formation of concretions in the oral cavity and in the salivary glands and ducts. C. Naeslund. Dental Cosmos, 1926, lxviii, 1137.

Plastic closure of an oral fistula of the maxillary antrum. J. Zange. Beitr. z. Anat., Physiol., Path. u. Therap. d. Ohres etc., 1926, xxiii, 685.

Pyorrhœa alveolaris and gastric acidity. Z. Sagal. Dental Cosmos, 1926, lxviii, 1145.

A non-operative method of treating alveolar pyorrhœa. F. Goldstein and E. Bachrach. Ztschr. f. Stomatol., 1926, xxiv, 542.

Epuloids in an infant 9 days old. A. Cardia. Ann. ital. di chir., 1926, v, 691.

Organic disturbances of dental origin. G. Di Domenico. Rassegna internaz. di clin. e terap., 1926, vii, 613.

Odontogenic septicæmias: retromaxillary adenophlegmon. J. Arís. Rev. méd. de Barcelona, 1926, iii, 292.

Histological findings in odontomata. H. Wolf. Ztschr. f. Stomatol., 1926, xxiv, 573.

The tongue in diagnosis. J. R. Irwin. South. M. & S., 1926, lxxxviii, 793.

A case of acute glossitis. N. M. Dave. Indian M. Gaz., 1926, lxi, 600.

Medio-lingual abscess. Tedenat. Med. Press, 1926, n. s. cxxii, 444.

Actinomycosis of the tongue: report of twelve cases. F. A. Figi. Surg. Clin. N. Am., 1926, vi, 1343. [265]

Leptothrix mycosis of the tongue. E. Roedelius and E. Brack. Arch. f. klin. Chir., 1926, cxli, 1.

Papilloma of the tongue. L. Zagni. Riforma med., 1926, xlii, 871.

The classification of oral tumors. C. W. Waldron. Am. J. Surg., 1926, n. s. i, 365.

Leucoplakia and cancer of the tongue. C. Villarán. Crón. méd., Lima, 1926, xlii, 189.

Certain problems in the early diagnosis of cancer of the mouth region. F. M. Johnson. J. Med. Ass. Georgia, 1926, xv, 453.

Radium and electrothermic methods in the treatment of lesions of the oral cavity. G. E. Ward. Internat. J. Med. & Surg., 1926, xxxix, 475.

Pharynx

Pharyngeal sinus with cervical Pott's disease; report of six cases. G. B. New and W. J. Decker. Surg. Clin. N. Am., 1926, vi, 1335. [265]

An anatomical study of the superficial and deep lymphoid tissues of the nose and throat. C. A. Crumrine Atlantic M. J., 1926, xxx, 58.

Cartilaginous or intratonsillar tissue. S. E. Parodi and H. Zubizarreta. Semana méd., 1926, xxxiii, 982.

The tonsils from the standpoint of the internist. G. M. Carlisle. Texas State J. M., 1926, xxii, 513.

Non-endoscopic cases of foreign bodies in the tonsil. L. H. Clerf. Arch. Otolaryngol., 1926, iv, 489.

Chronic focal infection of the tonsils and accessory sinuses in adults. J. Daland. Ann. Otol., Rhinol. & Laryngol., 1926, xxxv, 1064.

Giant urticaria due to diseased (pus) tonsils. S. Floersheim. California & West. Med., 1926, xxv, 751.

Irradiation of diseased tonsils. J. C. Scal. Med. J. & Rec., 1926, cxxiv, 673.

A bloodless tonsillectomy. R. L. Rosenfield. Med. J. Australia, 1926, ii, 861.

Tonsillectomy: a plea for the use of a clamp guillotine. J. S. Davis. Med. J. Australia, 1926, ii, 861.

Angioma of the tonsil. F. C. Ormerod. J. Laryngol. & Otol., 1926, xli, 797.

Neck

Two cases of suicidal cut-throat. S. Smith. Lancet, 1926, cxi, 1163.

A needle in the cervical region tolerated for five months. J. N. Roy. Arch. Otolaryngol., 1926, iv, 520.

The etiology of wry neck. H. Lichtenstein. Zentralbl. f. Chir., 1926, liii, 1185.

The so-called branchiogenetic carcinoma: its occupational incidence and origin. R. V. Hudson. Brit. J. Surg., 1926, xiv, 280. [265]

Fibrosarcoma of the neck: exeresis: immediate application of superheated air. Péraire. Bull. et mém. Soc. d. chirurgiens de Par., 1926, xviii, 602.

Parastruma. Miller. Zentralbl. f. allg. Path. u. path. Anat., 1926, xxxvii, 291.

Report of a case of cyst of the thyroglossal duct. E. G. GILL. Virginia M. Month, 1926, liii, 591.

Guanadine and the parathyroids. W. SUSMAN. Endocrinology, 1926, x, 445.

Infantile tetany. J. P. SCOTT and S. J. USHER. J. Am. M. Ass., 1926, lxxxvii, 1904.

Tuberculosis of the thyroid gland. F. A. COLLER and C. B. HUGGINS. Ann. Surg., 1926, lxxxiv, 804.

Some recent advances in clinical metabolimetry. R. V. BARBERA. Siglo méd., 1926, lxxiii, 319, 343, 367, 391.

Racial factor in basal metabolism. E. L. TURNER. J. Am. M. Ass., 1926, lxxxvii, 2052.

Hypothyroidism as a complication of diabetes mellitus. A. H. ROWE. Endocrinology, 1926, x, 499.

Some neglected factors in the goiter problem. M. A. AUSTIN. J. Indiana State M. Ass., 1926, xix, 478.

Isolated lateral accessory goiters. K. VOGEL. Ztschr. f. Hals-, Nasen-u. Ohrenheilk., 1926, xiv, 463.

The heart in thyroid disease. E. P. BOAS. Med. J. & Rec., 1926, cxxiv, 695.

Psychoses associated with goiter. C. E. BOYS. J. Michigan State M. Soc., 1926, xxv, 653.

Purulent strumitis following grippe. K. SCHULTZE-JENA. Zentralbl. f. Chir., 1926, liii, 2258.

Thyroid disturbances: a clinicopathological study of 300 instances. F. B. MENNE, T. M. JOYCE, and A. P. VON HUNGEN. Arch. Surg., 1926, xiii, 329. **[266]**

Goiter prophylaxis with iodine salt. B. BREITNER. Wien. klin. Wchnschr., 1926, xxxix, 42.

Iodine therapy in diseases of the thyroid gland. F. B. UTLEY. Atlantic M. J., 1926, xxx, 149.

The incorrect use of iodine in the treatment of goiter. D. GUTHRIE. Therap. Gaz., 1926, 3 s. xlii, 837.

Goiter: from the standpoint of prevention. W. D. KEITH. Canadian M. Ass. J., 1926, xvi, 1171. **[267]**

Goiter: its medical aspect. A. H. GORDON. Canadian M. Ass. J., 1926, xvi, 1176. **[267]**

Goiter: from a radiologist's viewpoint. W. H. MCGUFFIN. Canadian M. Ass. J., 1926, xvi, 1182. **[267]**

Goiter: Its surgical treatment. G. S. FAHRNI. Canadian M. Ass. J., 1926, xvi, 1188. **[267]**

The basal metabolism and the blood chemistry. J. M. MAYHEW Nebraska State M. J., 1926, xi, 409. **[268]**

The pathology of goiter. C. EMERSON. Nebraska State M. J., 1926, xi, 411. **[268]**

The medical management of goiter. R. W. BLISS. Nebraska State M. J., 1926, xi, 416. **[268]**

The X-ray treatment of goiter. E. W. ROWE. Nebraska State M. J., 1926, xi, 419. **[268]**

The diagnosis of toxic goiter. H. J. VANDEN BERG. J. Michigan State M. Soc., 1926, xxv, 656.

The management of patients with goiter. W. E. SISTRUNK. Minnesota Med., 1926, ix, 667.

Exophthalmic goiter. BÉRARD. Lyon chir., 1926, xxiii, 599.

Exophthalmic goiter and toxic adenoma. A. GRAHAM and E. C. CUTLER. Ann. Surg., 1926, lxxxiv, 497.

Modern management of exophthalmic goiter. J. DEJ. PEMBERTON. California & West. Med., 1926, xxv, 619.

The operative treatment of Basedow's disease. G. HOTZ. Deutsche med. Wchnschr., 1926, lii, 604 **[268]**

Radiation therapy of malignant goiter. C. BARTHELS. Med. Klin., 1926, xxii, 1364.

Local anæsthesia in toxic goiter. T. L. CHAPMAN. Minnesota Med., 1926, ix, 671.

Recognition of the goiter patient unsuited to thyroidectomy. W. BARTLETT. J. Am. M. Ass., 1926, lxxxvii, 1279.

Substernal goiter. F. H. LAHEY. J. Am. M. Ass., 1926, lxxxvii, 1282.

Thyroid surgery in America. W. S. HANDLEY. Lancet, 1926, ccxi, 1253.

The influence of the removal of the thyroid, parathyroid, and sexual glands and of thyroid feeding upon the regulation of the body temperature of rabbits. V. KORENCHEVSKY. J. Path. & Bacteriol., 1926, xxix, 461. **[269]**

Goiter operation without direct ligation of the thyroid arteries. J. L. MUELLER. Muenchen. med. Wchnschr., 1926, lxxiii, 954.

Sudden death after strumectomy in a series of 1,000 goiter operations. H. DOERFLER. Beitr. z. klin. Chir., 1926, cxxxvii, 465.

Hæmorrhage after goiter operation as vicarious menstruation. JALCOWITZ. Deutsche Ztschr. f. Chir., 1926, cxcviii, 101.

Thyroid cancer after intervention for goiter. BÉRARD. Lyon chir., 1926, xxiii, 645.

On the interpretation of speech sounds by means of their tactual correlates. R. H. GAULT. Ann. Otol., Rhinol. & Laryngol., 1926, xxxv, 1050.

Defects of speech. W. J. PEPARD. Laryngoscope, 1926, xxxvi, 892.

Congenital laryngeal obstruction, with the report of a case. F. D. GUNN. Bull. Buffalo Gen. Hosp., Buffalo, N. York, 1926, iv, 46.

Payr's laryngoplasty and paralysis of the recurrent nerve. V. SCHMIEDEN. Muenchen. med. Wchnschr., 1926, lxxiii, 558.

Laryngeal and œsophageal sequelæ of diphtheria. L. H. CLERF. Laryngoscope, 1926, xxxvi, 912.

Membranous laryngitis and tracheobronchitis (non-diphtheritic). T. R. GITTINS. Ann. Otol., Rhinol. & Laryngol., 1926, xxxv, 1110.

Alterations of laryngeal motility in pulmonary tuberculosis not complicated by laryngeal tuberculosis. E. RICCITELLI and Y. FRANCHINI. Semana méd., 1926, xxxiii, 1023.

Tuberculosis of the throat. G. B. WOOD. Arch. Otolaryngol., 1926, iv, 527.

The diagnosis of early tuberculous laryngitis. S. HANDS. J. Iowa State M. Soc., 1926, xvi, 529.

The diagnosis and treatment of laryngeal tuberculosis. E. A. LOOPER. Ann. Otol., Rhinol. & Laryngol. 1926, xxxv, 1041.

Carcinoma of larynx, pharynx, trachea, and œsophagus. F. J. TOREK. Ann. Surg., 1926, lxxxiv, 889.

Biopsy in suspicious (carcinomatous) growths of the larynx. C. J. IMPERATORI. Ann. Otol., Rhinol. & Laryngol., 1926, xxxv, 1241.

SURGERY OF THE NERVOUS SYSTEM

Brain and Its Coverings; Cranial Nerves

Acute craniocerebral injuries. O. S. WAUGH. Canadian M. Ass. J., 1926, xvi, 1475.

Head injuries marked by intracranial tension. F. W. PARHAM. New Orleans M. & S. J., 1926, lxxix, 377.

Living dogs whose skulls were subjected to roentgen irradiation. DEMEL. Zentralbl. f. Chir., 1926, liii, 2155. **[270]**

A clinical contribution upon late posttraumatic apoplexy. A. WINKELBAUER. Deutsche Ztschr. f. Chir., 1926, cxcvi, 1.

Ventricular puncture in apoplexy. H. CARLILL. Lancet, 1926, ccxi, 1212.

A head rest for cerebellar exploration. CALDWELL. Ann. Surg., 1926, lxxxiv, 894.

Cerebroventriculomyelography with conclusions; an historical sketch. D. G. DICKERSON. Northwest Med., 1926, xxv, 642.

Cerebral localization. Lancet, 1926, ccxi, 1217.

Brain cyst. A. A. MATHEWS. Northwest Med., 1926, xxv, 640.

Brain abscess. C. A. ELSBERG. Ann. Surg., 1926, lxxxiv, 886.

Abscesses of the left frontal and temporal lobes of metastatic origin. P. L. MAHONEY. Laryngoscope, 1926, xxxvi, 898.

The diagnosis and treatment of brain tumors. W. E. DANDY. Northwest Med., 1926, xxv, 629.

A clinical study of intracranial tumors and especially of some errors in their diagnosis. Sir J. PURVES-STEWART. Proc. Roy. Soc. Med., Lond., 1926, xx, Sect. Neurol., 1.

Types of cerebral tumors. P. SARGENT. Brit. M. J., 1926, ii, 628. [270]

A new form of craniotome for opening the skull. H. S. SOUTTAR. Brit. M. J., 1926, ii, 630. [270]

Localization by the X-rays. A. P. BERTWISTLE. Brit. M. J., 1926, ii, 631. [270]

Clinical experiences with the development and removability of tumors at the cerebellopontine angle on the basis of twenty-two observations. E. HEYMANN. Beitr. z. klin. Chir., 1926, cxxxvi, 385. [271]

A tumor of the medulla oblongata. R. LOZANO and I. COSTERO. Deutsche Ztschr. f. Chir., 1926, cxcviii, 270.

Multiple metastatic tumors of the brain from a primary melanosarcoma of the neck. H. L. MITCHELL. Atlantic M. J., 1926, xxx, 162.

The functions of the hypophysis and the infundibulohypothalamic region. B. A. HOUSSAY. Rev. méd. de Barcelona, 1926, iii, 328.

The action of hypophyseal preparations upon basal metabolism. M. R. CASTEX and M. SCHTEINGART. Rev. Soc. argent. de biol., 1926, ii, 379.

Basal metabolism in hypophyseal conditions. M. R. CASTEX and M. SCHTEINGART. Arch. argent. de enferm. d. apar. digest., 1926, ii, 53.

Pituitary disease. J. S. LANKFORD. Clin. Med., 1926, xxxiii, 860.

Hypophysectomy and replacement therapy in relation to basal metabolism and specific dynamic action in the rat. G. L. FOSTER and P. E. SMITH. J. Am. M. Ass., 1926, lxxxvii, 2151.

Pituitary tumor on the floor of the third ventricle. C. A. ELSBERG. Ann. Surg., 1926, lxxxiv, 888.

Spontaneous temporoparietal pneumatocele of mastoid origin. GABRIELLE. Lyon chir., 1926, xxiii, 655.

Twenty years of brain surgery. V. MAGNUS. Zentralbl. f. Chir., 1926, liii, 1149.

Experiences in brain surgery. H. OLIVECRONA. Zentralbl. f. chir., 1926, liii, 1149.

Epidemic meningitis. J. B. NEAL, H. W. JACKSON, and E. APPELBAUM. J. Am. M. Ass., 1926, lxxxvii, 1992.

Cystic serous meningitis. G. J. JENKINS. J. Laryngol. & Otol., 1926, xli, 781.

Otitic meningitis. W. P. EAGLETON. J. Am. M. Ass., 1926, lxxxvii, 1544. [271]

The chemistry of the cerebrospinal fluid in otitic meningitis. J. G. GREENFIELD. J. Laryngol. & Otol., 1926, xli, 785.

The value of examination of the cerebrospinal fluid in the diagnosis of tuberculous meningitis. F. DEP. RIVAS MAZA. Rev. de med. y cirug., Caracas, 1926, ix, 117.

An unusual case of tuberculous meningitis. R. W. BENTON. Rhode Island M. J., 1926, ix, 195.

A case of meningitis melitensis. DESAGE, PELLERIN and VIÑERTA. Prog. de la clin., Madrid, 1926, xiv, 452.

Actinobacillus meningitis. L. GERDINE and D. PEASE. Am. J. Dis. Child., 1926, xxxii, 878.

The treatment of trigeminal neuralgia with alcoholic injections. N. N. NASAROFF. Zentralbl. f. Chir., 1926, liii, 1946.

A periarterial sympathectomy in trigeminal neuralgia. A. W. WISCHNEWSKY. Zentralbl. f. Chir., 1926, liii, 1178.

Division of the sensory root on both sides. C. H. FRAZIER. J. Am. M. Ass., 1926, lxxxvii, 1730.

The technique of posterior ganglion resection in trigeminal neuralgia. H. A. SOELLING. Zentralbl. f. Chir., 1926, liii, 1150.

A chondroma involving the gasserian ganglion. C. A. ELSBERG. Ann. Surg., 1926, lxxxiv, 887.

Resection of the superior and inferior maxillary nerves at their exit from the cranium. AVELINO and GUTIERREZ. Rev. de cirug., Buenos Aires, 1926, v, 456.

Plastic operations for facial paralysis. H. BRUNNER. Arch. f. klin. Chir., 1926, cxl, 85. [272]

Erosion of the petrous bone by an acoustic nerve tumor: demonstration by the roentgen ray. E. B. TOWNE. Arch. Otolaryngol., 1926, iv, 515.

Spinal Cord and Its Coverings

Diagnostic intraspinal injections of lipiodol. SICARD and HAGUENEAU. Ann. Surg., 1926, lxxxiv, 894.

Hæmatic cysts of the cauda equina demonstrated by lipiodol examination; laminectomy; recovery. BÉRARD and FROMENT. Lyon chir., 1926, xxiii, 593.

The indications for Foerster's operation. A. KORTZEBORN. Zentralbl. f. Chir., 1926, liii, 1435.

Peripheral Nerves

An electrophysiological contribution on the metabolism of the peripheral nerves. C. VIALE. Rev. Soc. argent. de biol., 1926, ii, 523.

A contribution on the symptoms of nerve injuries. L. SEYBERTH. Zentralbl. f. Chir., 1926, liii, 1632.

An unusual case of neuritis. J. F. MALLOY. J.-Lancet, 1926, n. s. xlvi, 558.

Contractured ulnar paralysis relieved by treatment of the nasal (spheno-palatine) ganglion. W. R. BRANDON. Arch. Otolaryngol., 1926, iv, 493.

Phrenospasm in infancy: report of a case. R. M. TYSON. Arch. Pediat., 1926, xliii, 818.

Recklinghausen's disease. PATEL and DECHAUME. Lyon chir., 1926, xxiii, 631.

Cavernous neurofibrosarcoma of the brachial plexus. A. CEBALLOS. Crón. méd., Lima, 1926, xliii, 276.

Chronic neuralgia of the lowest intercostal nerves and its treatment. BAUM. Deutsche Ztschr. f. Chir., 1926, cxcvii, 74.

Injury of the ischiatic nerve through a typical movement complex. F. KAZDA. Arch. f. orthop. u. Unfall-Chir., 1926, xxiv, 233.

The nature and treatment of sciatica. A. E. HERTZLER. Am. J. Surg., 1926, n.s. i, 200. [272]

Sympathetic Nerves

The influence of cervical sympathectomy in autoplastic and hæmoplastic skin grafts. F. P. TINOZZI. Rassegna internaz. di clin. e terap., 1926, vii, 585.

The treatment of facial paralysis by resection of the cervical ganglion of the sympathetic. BOTREAU-ROUSSEL

and ROBINEAU. Bull. et mém. Soc. nat. de chir., 1926, lii, 896.

Accessory nerves in the thorax in man. N. S. KONDRATJEW. Anat. Anz., 1926, lxi, 69.

The surgery of the cervical, thoracic, and abdominal sympathectic system with particular reference to the surgical treatment of bronchial asthma and angina pectoris. E. HESSE. Beitr. z. klin. Chir., 1926, cxxxvi, 429.

The surgical treatment of bronchial asthma. H. FREUND. Beitr. z. klin. Chir., 1926, cxxxvi, 581.

The causes of failure in operations for asthma and their prevention. KUEMMELL. Zentralbl. f. Chir., 1926, liii, 1278. [273]

Injury of the heart muscle after division of the extracardial nerves, and a contribution on the operative treatment of angina pectoris. H. ISAKOWITZ. Zentralbl. f. Herz.- u. Gefaesskrankh., 1926, xviii, 105. [273]

Experiences with regard to the efficiency of the vagus after bilateral extirpation of the stellate ganglion. R. DITTLER and H. K. MUELLER. Ztschr. f. d. ges. exper. Med., 1926, l, 166. [273]

The report of the result a year after a lumbar ramicotomy for Little's disease. R. LERICHE. Lyon chir., 1926, xxiii, 666.

Ramisection of the lumbar and sacral sympathetic. G. PIERI. Ann. ital. di chir., 1926, v, 639.

The electrocardiogram in unilateral and bilateral sympathectomy. M. MANDELSTAMM. Beitr. z. klin. Chir., 1926, cxxxvi, 499.

Periarterial sympathectomy. J. W. VAUGHAN. Ann. Clin. Med., 1926, v, 529.

Report of a case of periarterial sympathectomy with tubulization. M. SAIDMANN. Zentralbl. f. Chir., 1926, liii, 1112.

Brief considerations on an unusual case of periarterial sympathectomy. O. FINZI. Riforma med., 1926, xlii, 918.

Miscellaneous

Infection and the nervous system. H. F. O. HABERLAND. Muenchen. med. Wchnschr., 1926, lxxiii, 1389.

A new colloidal reaction of the cerebrospinal fluid. R. CALATRONI. Rev. Soc. argent. de biol., 1926, ii, 503.

The vacuum headache. C. W. JENNINGS. South. M. & S., 1926, lxxxviii, 787.

Epiloia. T. BRUSHFIELD and W. WYATT. Brit. J. Child, Dis., 1926, xxiii, 254.

The question of genuine epilepsy. TILMANN. Deutsche med. Wchnschr., 1926, lii, 1595.

On the relationship of epilepsy and the circulatory system. A. G. ODELL. Clifton M. Bull., Clifton Springs, N. York, 1926, xii, 138.

The cutaneous plantar reflex in cranial traumatisms. E. SANTORO. Ann. ital. di chir., 1926, v, 910.

The indications for cisterna puncture. S. S. BERGER and M. H. GROSSBERG. Ohio State M. J., 1926, xxii, 1039.

Studies in acromegaly. II. Historical note. L. M. DAVIDOFF. Endocrinology, 1926, x, 453.

Studies in acromegaly. III. The anamnesis and symptomatology in 100 cases. L. M. DAVIDOFF. Endocrinology, 1926, x, 461.

Multiple neurofibromatosis. V. TRAMONTANO and F. PANSINI. Rassegna internaz. di clin. e terap., 1926, vii, 513.

Trophoneurotic ulcer of the gluteal region developing after a shrapnel injury of the cauda equina. F. HAGEN. Muenchen. med. Wchnschr., 1926, lxxiii, 1395.

The control of pain through the nasal ganglion. J. E. LOFTUS. Therap. Gaz., 1926, 3 s. xlii, 847.

SURGERY OF THE CHEST

Chest Wall and Breast

A plastic operation for hanging breast. J. FRIST. Wien. med. Wchnschr., 1926, lxxvi, 617.

Primary tuberculosis of the mammary gland: a clinical and pathological study. P. LEONE. Arch. di ostet. e ginec., 1926, 2 s. xiii, 309.

Tuberculosis of the male breast. R. H. J. SWAN and H. J. B. FRY. Brit. J. Surg., 1926, xiv, 234. [274]

The electric capacity of tumors of the breast. H. FRICKE and S. MORSE. J. Cancer Research, 1926, x, 340.

Carcinoma of the breast. R. B. GREENOUGH. Am. J. Roentgenol., 1926, xvi, 439.

Regressive changes, fibromatosis, and cancer of the breast. A. ROSENBURG. Deutsche Ztschr. f. Chir., 1926, cxcviii, 130.

Stewart's incision in radical surgery of the breast. R. E. STONE. New Orleans M. & S. J., 1926, lxxxix, 417.

The treatment of cancer of the breast from the viewpoint of a radiologist. A. SOILAND. Brit. J. Radiol., 1926, xxxi, 469.

Cancer of the breast, a discussion of roentgen-ray technique in its treatment. I. L. MCGLASSON and C. F. LEHMAN. Texas State J. M., 1926, xxii, 528.

Preventive and postoperative radiotherapy in cancer of the breast. A. GUNSETT. Cancer, 1926, iii, 5.

The rationale and technique of combining operation with radium treatment in cancer. W. C. STEVENSON. Lancet, 1926, ccxi, 1158.

Postoperative treatment of cancer of the breast. H. W. CARSON. Brit. M. J., 1926, ii, 1095.

Carcinoma mammæ: the results of treatment. J. F. C. BRAINE and G. MASSIE. Guy's Hosp. Rep., Lond., 1926, lxxvi, 484.

Trachea, Lungs, and Pleura

Intubation and tracheotomy in diphtheria. A. J. WAGERS. Laryngoscope, 1926, xxxvi, 905.

On a method of introducing lipiodol in the bronchi for diagnostic purposes. F. ABRAMOVITCH. Brit. J. Radiol., 1926, xxxi, 487.

Limitations in the use of lipiodol in the diagnosis of diseases of the lungs. P. P. VINSON and W. S. LEMON. Med. Clin. N. Am., 1926, x, 553.

Bronchography following the "passive" introduction of contrast media into the tracheobronchial tree. A. OCHSNER. Wisconsin M. J., 1926, xxv, 544.

The solitary circumscribed round shadows in the lung roentgenogram. M. LUEDIN. Fortschr. a. d. Geb. d. Roentgenstrahlen, 1926, xxxiv, 899.

A case of pneumolith spontaneously expelled by coughing. T. G. BENJAMIN and W. M. MOLLISON. Guy's Hosp. Rep., Lond., 1926, lxxvi, 475.

A method of removing a bead from a bronchus. S. JESBERG. Laryngoscope, 1926, xxxvi, 917.

The results of ligation of the pulmonary arteries: an experimental study. S. A. SMIRNOFF. Arch. f. klin. Chir., 1926, cxli, 512.

On pathological conditions in the upper respiratory tract, and the importance of their recognition and correction in chest disabilities. W. J. BROWN. Canadian M. Ass. J., 1926, xvi, 1480.

An attempt to appraise the roentgenological method of diagnosis of pulmonary tuberculosis. F. S. BISSELL. Am. J. Roentgenol., 1926, xvi, 544.

The surgical treatment of pulmonary tuberculosis. F. P. MILLER. Texas State J. M., 1926, xxii, 507.

Iodized oil with reference to bronchiectasis. B. WHYTE. Canadian M. Ass. J., 1926, xvi, 1443.

Bronchiectasis in childhood. W. S. LEMON. Med. Clin. N. Am., 1926, x, 531.

Three cases of bronchiectasis. W. P. H. SHELDON and H. THURSFIELD. Proc. Roy. Soc. Med., Lond., 1926, xx, 79.

Resolution in pneumonic consolidations. R. G. ALLISON. Am. J. Roentgenol., 1926, xvi, 549.

Non-tuberculous pulmonary suppuration cured after pleuroparietal detachment: result after three years. P. MOIROUD and J. L. ROUX-BERGER. Bull. et mém. Soc. nat. de chir., 1926, lii, 861.

Artificial pneumothorax, personal experience in its use. R. A. PEERS. Northwest Med., 1926, xxv, 651.

Home treatment of tuberculosis by artificial pneumothorax. M. J. FINE. J. Med. Soc. N. Jersey, 1926, xxiii, 614.

Different clinical and roentgenological aspects of therapeutic pneumothorax. E. LEURET, G. AUMONT, and J. CAUSSIMON. J. de méd. de Bordeaux, 1926, ciii, 779.

Remarks on the surgical treatment of pulmonary tuberculosis. H. ALEXANDER. Beitr. z. Klin. d. Tuberk., 1926, lxiii, 388.

Severe complications in the surgical treatment of tuberculosis and the simultaneous employment of exeresis of the phrenic nerve and pneumothorax. H. DEIST. Beitr. z. Klin. d. Tuberk., 1926, lxiii, 424.

The reaction of the pleura and lungs to roentgen rays. A. U. DESJARDINS. Am. J. Roentgenol., 1926, xvi, 444.

Spontaneous pneumothorax. A. W. ATWOOD. Boston M. & S. J., 1926, cxcv, 1237.

Spontaneous pneumothorax—with a report of cases. H. B. MULHOLLAND and J. C. FLIPPIN. Virginia M. Month., 1926, liii, 565.

The production of artificial pleural adhesions: an experimental study. R. B. BETTMAN. Surg., Gynec. & Obst., 1926, xliii, 599.

Elastic restraint for fibrinous pleurisy. L. DUNN. J. Am. M. Ass., 1926, lxxxvii, 1996.

Chronic bilateral pleural effusion and mediastinitis. W. H. SWAN and L. W. BORTREE. J. Am. M. Ass., 1926, lxxxvii, 2162.

A method of manual pleuropneumolysis for the production of therapeutic pneumothorax in cases of pleural symphysis. D. PATERNOSTER. Riforma med., 1926, xlii, 918.

Advantages of the closed method of treating empyema. R. B. BETTMAN. Illinois M. J., 1926, l, 501.

Persistent empyema cavities and their treatment. BERNHOLZ. Med. Klin., 1926, xxii, 1450.

Endothelioma of the diaphragmatic and mediastinal pleura. P. ESCUDERO and J. W. TOBÍAS. Semana méd., 1926, xxxiii, 932.

Primary endothelioma of the diaphragmatic pleura; the phrenicoparietal syndrome. G. A. ALFARO and P. COSSIO. Semana méd., 1926, xxxiii, 1045.

Radical chest surgery in emergency cases. D. MACRAE, JR. Med. Herald & Physiotherap., 1926, xlv, 317.

Heart and Pericardium

Gunshot and stab wounds of the heart. ROTHFUCHS. Deutsche Ztschr. f. d. ges. gerichtl. Med., 1926, viii, 151.

Two cases of wounds transfixing the heart. OUDARD. Bull. et mém. Soc. nat. de chir., 1926, lii, 872.

Report of a case of coronary embolism with rupture of the heart. W. F. BOPP. N. York State J. M., 1926, xxvi, 977.

Experimental studies of the pain sensibility of the heart and the great vessels and their relation to angina pectoris. R. SINGER. Wien. Arch. f. inn. Med., 1926, xiii, 157.

Œsophagus and Mediastinum

A case of double twist without stricture of the terminal portion of the œsophagus. H. P. MOSHER. Ann. Otol., Rhinol. & Laryngol., 1926, xxxv, 969.

Foreign body complications of lye stricture of the œsophagus. L. RICHARDS. Boston M. & S. J., 1926, cxcv, 1108.

The Plummer-Vinson syndrome (spasm of the pharyngo-œsophageal sphincter with anæmia and splenomegaly). A. F. HURST. Guy's Hosp. Rep., Lond., 1926, lxxvi, 426.

A case of fatal rupture of the cardia in dilatation for cardiospasm with Stark's sound. V. MAYDL. Med. Klin., 1926, xxii, 408. [275]

Emphysema of the skin after œsophagoscopy. J. ZIPPER. Deutsche Ztschr. f. Chir., 1926, cxcvi, 223.

The principles of the treatment of œsophageal tuberculosis with particular reference to the histology of the tubercle and the pathology of surgical tuberculosis. K. MÉSZÁROS. Arch. f. klin. Chir., 1926, cxli, 170.

The dermatojejunoplastic operation on the œsophagus. P. ALBRECHT. Zentralbl. f. Chir., 1926, liii, 1692.

Hodgkin's disease primary in the thymus gland; report of a case in an infant. M. WOLLSTEIN and S. MCLEAN. Am. J. Dis. Child., 1926, xxxii, 889.

Primary carcinoma of the thymus. I. I. LEMANN and J. SMITH. Arch. Int. Med., 1926, xxxviii, 807.

Spirochætæ in the thymus gland: a case report. J. R. MCCORD. Am. J. Obst. & Gynec., 1926, xii, 890.

The roentgenological differential diagnosis of aortic aneurisms and mediastinal tumors. R. KIENBOECK. Fortschr. a. d. Geb. d. Roentgenstrahlen, 1926, xxxiv, 849.

Mediastinal tumor or aortic aneurism: an interesting case from the standpoint of differential diagnosis. O. HOCHE. Deutsche Ztschr. f. Chir., 1926, cxcvi, 219.

Acute suppurative mediastinitis: report of a case. F. E. BALL. Arch. Otolaryngol., 1926, iv, 512.

Dermoid cysts of the anterior mediastinum. E. NICHOLSON and J. F. TOURREILLES. Semana méd., 1926, xxxiii, 1142.

Miscellaneous

Diseases of the chest demonstrated by lipiodol. A. H. PIRIE. Am. J. Roentgenol., 1926, xvi, 553.

SURGERY OF THE ABDOMEN

Abdominal Wall and Peritoneum

A case of ossification of an abdominal cicatrix. P. MORNARD. Bull. et mém. Soc. d. chirurgiens de Par., 1926, xviii, 596.

A discussion of postoperative gas bacillus infection of the abdominal wall. D. D. BUTLER. Ann. Surg., 1926, lxxxiv, 841.

Hernia. W. P. FITE. J. Oklahoma State M. Ass., 1926, xix, 325.

Umbilical hernia and abdominal fissure. WILDEGANS. Zentralbl. f. Chir., 1926, liii, 2277.

X-ray findings in a large ventral hernia. J. E. HABBE. Radiology, 1926, vii, 511.

Sliding hernia of the large intestine. A. JACOBSON. Arch. f. klin. Chir., 1926, cxli, 286.

Hernia of the semilunar line of Spigelius. P. MEYER. Zentralbl. f. Chir., 1926, liii, 1949.

A curious case of giant lumbar hernia. R. J. ROSÉS, A. B. SERRANO, I. FIGUERAS, and F. FAIXAT. Rev. méd. de Barcelona, 1926, iii, 332.

Right incarcerated juxtavesical hernia. N. BELU. Zentralbl. f. Chir., 1926, liii, 1124.

An acute condition of the abdomen in a nursing baby with symptoms of hernia strangulation. J. S. GONZÁLEZ and A. JOHOW. Bol. Soc. de cirug. de Chile, 1926, iv, 176.

False hernial strangulation associated with suppurative peritonitis. C. OLIVA. Policlin., Rome, 1926, xxxiii, sez. chir., 468.

Operative cure of a severe incarcerated hernia in a child 14 days old. H. PRAETORIUS. Med. Klin., 1926, xxii, 1452.

A contribution on the plastic closure of herniæ through the uterus. F. C. HILGENBERG. Beitr. z. klin. Chir., 1926, cxxxvii, 173.

Anæsthesia in inguinal hernia. S. OSTROWSKI. Muenchen. med. Wchnschr., 1926, lxxiii, 950.

Inguinal hernioplasty. L. F. WATSON. Am. J. Surg., 1926, n.s. i, 392.

The absorption of bacteria from the abdominal cavity. M. P. SSOKOLOVSKIJ. Vestnik chirurgii i pogranicnych oblastej, 1925, iv, 20. **[276]**

The treatment of suppurative peritonitis. R. PAMPERL. Med. Klin., 1926, xxii, 1409, 1446.

The treatment of suppurative diffuse peritonitis. KIRSCHNER. 50 Tag. d. deutsch. Ges. f. Chir., Berlin, 1926. **[276]**

The action of oxygen in exudative tuberculous peritonitis. D. MAESTRINI. Riforma med., 1926, xlii, 892.

The optimal hydrogen-ion concentration as the most important factor in the disinfection of local and general infections and its value in the treatment of purulent inflammations of the peritoneum. F. KEYSSER and O. ORNSTEIN. Klin. Wchnschr., 1926, v, 404.

Clinical and experimental contributions upon foreign body tuberculosis of the peritoneum. C. CORONINI and S. IATROU. Deutsche Ztschr. f. Chir., 1926, cxcviii, 137.

Metastatic peritonitis. R. GIRGENSOHN. Arch. f. klin. Chir., 1926, cxli, 196.

Pseudomyxoma peritonei and its metastasis in a patient. H. HAMDI. Zentralbl. f. allg. Path. u path. Anat., 1926, xxxvii, 459.

Free edges of omentum in the curative and prophylactic treatment of postoperative intraperitoneal adhesions. A. CEBALLOS. Arch. argent. de enferm. d. apar. digest., 1926, ii, 5.

Late disturbances from gangrene of previously incarcerated omentum. E. METGE. Zentralbl. f. Chir., 1926, liii, 1574.

A case of hæmaturia relieved after removal of a cystic tumor of the great omentum. B. S. RAU. Indian M. Gaz., 1926, lxi, 599.

Mesenteric lymphangeitis. B. O. PRIBRAM. Arch. f. klin. Chir., 1926, cxl, 589. **[281]**

Solid tumor of the mesentery. P. BONNET. Lyon chir., 1926, xxiii, 596.

Gastro-Intestinal Tract

Foreign bodies in the alimentary tract. W. C. SHIPLEY. California & West. Med., 1926, xxv, 751.

Radiological study of the digestive tract in normal infants. DE BACKER and VAN DE PUTTE. Brit. J. Radiol., 1926, xxxi, 493.

Metallic foreign bodies in the stomach. MORNARD and GOLDMAN. Bull. et. mém. Soc. d. chirurgiens de Par., 1926, xviii, 604.

Congenital abnormalities of the stomach, with the report of a rare case. D. A. RHINEHART and B. A. RHINEHART. Radiology, 1926, vii, 492.

Gastroptosis. T. MARTINI. Semana méd., 1926, xxxiii, 1118.

Partial gastric volvulus in a case of displacement of the stomach through a diaphragmatic defect. H. HABERER. Deutsche Ztschr. f. Chir., 1926, cxcv, 80.

Acquired pseudo-diverticulum of the stomach. E. VON KOENTZEY. Zentralbl. f. Chir., 1926, liii, 2265.

Ten cases of cardiospasm treated by Plummer's method of dilatation. N. AAGE-NIELSEN. Zentralbl. f. Chir., 1926, liii, 1144.

The operative treatment of true cardiospasm. E. CRONE-MUENZEBROCK. Zentralbl. f. Chir., 1926, liii, 2386.

Cardio-œsophageal relaxation. S. A. ROBINS and I. R. JANKELSON. J. Am. M. Ass., 1926, lxxxvii, 1961.

Studies of the pyloroduodenal juncture and the human duodenum. F. SPATH. Deutsche Ztschr. f. Chir., 1926, cxcvi, 39.

"Cyclic vomiting" in infants and children, including an analysis of the occurrence of vomiting in 200 children examined roentgenologically. L. T. LEWALD. Radiology, 1926, vii, 410.

Pyloric stenosis. A. S. GOUDIE. Brit. M. J., 1926, ii, 987.

Stenosis of the pylorus. DEMEL. Zentralbl. f. Chir., 1926, liii, 1698.

Familial incidence of pyloric stenosis. E. J. CAULFIELD. Am. J. Dis. Child., 1926, xxxii, 706.

Pyloric stenosis in brothers. J. W. HEEKES. Brit. M. J., 1926, ii, 835.

The operative treatment of pyloric stenosis. SCHULTE. Monatsschr. f. Kinderh., 1926, xxxii, 243.

The present status of gastroscopy. A. HUEBNER. Deutsche med. Wchnschr., 1926, lii, 1123.

The present status of gastroscopy. H. HOHLWEG. Karlsbader aerztl. Vortraege, 1926, vii, 150.

Recent advances in the physiology of gastric and pancreatic secretion. A. C. IVY. Northwest Med., 1926, xxv, 589.

Examination of the secretory function of the stomach. A. GOVAERTS and M. CORNIL. Bruxelles-méd., 1926, vi, 1545.

Fractional emptying of the stomach as a differential diagnostic measure. K. TIEFENSEE. Muenchen. med. Wchnschr., 1926, lxxiii, 1401.

A new method of gastric analysis. A. M. ALTSHULER. Ann. Clin. Med., 1926, v, 464.

A quantitative study of the effect of sodium bicarbonate on gastric function. C. S. KEEFER and A. L. BLOOMFIELD. Bull. Johns Hopkins Hosp., Balt., 1926, xxxix, 379.

Fractional gastric analysis with histamine. A. F. R. ANDRESEN. Ann. Clin. Med., 1926, v, 472.

Dyspepsia. H. H. BULLMORE. Med. J. Australia, 1926, ii, 836.

Chromoscopy of the stomach. K. GLAESSNER. Med. Klin., 1926, xxii, 887.

Gastric radioscopy and its interpretation. M. C. GUAS. Noticias med., 1926, i, 25.

Lesions of the stomach and duodenum. D. C. BALFOUR. Surg. Clin. N. Am., 1926, vi, 1175.

Cryptogenetic gastrorrhagia: an anatomicopathological study of the stomach. J. B. LLOSA and P. A. SAFFORES Semana méd., 1926, xxxiii, 1149.

The question of parenchymatous gastric hæmorrhage. H. PUHL. Deutsche Ztschr. f. Chir., 1926, cxcvii, 383.

A contribution on spurious melæna. U. FRANKE. Deutsche med. Wchnschr., 1926, lii, 993.

Gastric ulcer. C. J. MACGUIRE, JR. Ann. Surg., 1926, lxxxiv, 891.

The diagnostic value of disturbances of the acid-base equilibrium of the organism in gastric and duodenal ulcer. H. L. POPPER. Wien. klin. Wchnschr., 1926, xxxix, 1017.

The ulcer patient: studies of the constitution and general symptoms in chronic peptic ulcer, with particular reference to the vegetative nervous system. W. RUHMANN. Arch. f. Verdauungskr., 1926, p. 1.

The importance of the structure and function of the uterine mucous membrane in the development of chronic gastric ulcer. F. BUECHNER and C. RUF. Frankfurt. Ztschr. f. Path., 1926, xxxiii, 406.

The mechanism of pain in gastric and in duodenal ulcer: II. The production of pain by means of chemical irritants. W. L. PALMER. Arch. Int. Med., 1926, xxxviii, 694.

A case of conjugate ulcers of the pylorus and of the lesser curvature. J. R. GOYENA. Semana méd., 1926, xxxiii, 977.

The diagnosis of ulcer perforation: subcutaneous emphysema as a symptom of perforation. J. VIGYÁZÓ. Zentralbl. f. Chir., 1926, liii, 1627.

A case of penetrating gastric ulcer healed medically. M. B. KUNSTLER. Med. J. & Rec., 1926, cxxiv, 756.

Some problems of peptic ulcer. K. L. THORSGAARD. Clin. Med., 1926, xxxiii, 851.

The treatment of gastric ulcer. F. KOENIG. Muenchen. med. Wchnschr., 1926, lxxiii, 51. [281]

The medical treatment of non-complicated gastric ulcer. F. DEP. RIVAS MAZA. Rev. de med. y cirug., Caracas, 1926, ix, 123.

The treatment of organic hourglass stomach. E. UNGER and S. GLASSCHEIB. Arch. f. Verdauungskrankh., 1926, xxxvii, 371.

Postoperative hæmorrhage after gastro-enterostomy. M. KRABBEL. Zentralbl. f. Chir., 1926, liii, 1170.

Inflammatory stenosis of the gastro-enterostomy opening. DELORE. Lyon chir., 1926, xxiii, 591.

When is resection indicated in perforated gastric and duodenal ulcer? L. DRUENER. Zentralbl. f. Chir., 1926, liii, 1310.

Palliative resection in non-resectable ulcer of the stomach and duodenum. SCHOMBERG. Zentralbl. f. Chir., 1926, liii, 2353.

Experiences with the resection method of Pólya in ulcer of the stomach and duodenum. E. LINDBOE. Zentralbl. f. Chir., 1926, liii, 1142. [282]

A case of recurring ulcer after the Billroth I operation. H. BOERGER. Zentralbl. f. Chir., 1926, liii, 1499.

A case of leiomyoma of the stomach. J. MARTÍNEZ and T. C. MINNHAAR. Rev. Soc. argent. de biol., 1926, ii, 518.

Early diagnosis of gastric cancer. H. B. DEVINE. J. Cancer, 1926, iii, No. 12, 27.

Diffuse infiltration of the stomach due to a colloid carcinoma; colloid degeneration of the peritoneum. C. B. UDAONDO and J. E. CARULLA. Arch. argent. de enferm. d. apar. digest., 1926, ii, 39.

Ulcer carcinoma of the stomach. C. TAGE-HANSEN. Zentralbl. f. Chir., 1926, liii, 1144.

The frequency of ulcer carcinoma and the resulting indications for operative treatment. REISCHAUER. Zentralbl. f. Chir., 1926, liii, 1216.

A long-continued surgical cure of gastric cancer. C. WEGELE. Muenchen. med. Wchnschr., 1926, lxxiii, 819.

A system of general and special surgery: M. KIRSCHNER and A. NORDMANN. Part X, Vol. 5. Surgery of the stomach and duodenum: N. GULEKE, H. NIEDE, and H. SMIDT. 1926: Berlin, Urban & Schwarzenberg.

Bacteriological studies in relation to gastric surgery. W. LOEHR. Zentralbl. f. Chir., 1926, liii, 1618.

The internal use of presojod in gastric surgery. C. HAMMESFAHR. Zentralbl. f. Chir., 1926, liii, 1180.

Pyloromyotomy by Payr's method. E. STEDEN. Zentralbl. f. Chir., 1926, liii, 2353.

A case of high grade gastric stenosis after transverse resection of the stomach. P. WALZEL. Deutsche Ztschr. f. Chir., 1926, cxcvi, 228.

The question of drainage of the abdominal cavity after gastric resection. H. FINSTERER. Wien. med. Wchnschr., 1926, lxxvi, 599.

The treatment of postoperative gastric hæmorrhages with injections of the patient's blood. C. RAUSCHE. Deutsche Ztschr. f. Chir., 1926, cxcviii, 108.

The effect of bacillus acidophilus on intestinal putrefaction and the indican output. M. KAHN. J. Lab. & Clin. Med., 1926, xii, 226.

Intestinal parasitism. J. F. GONZÁLEZ. An. Fac. de med., Univ. de Montevideo, 1926, xi, 462.

Intestinal parasitosis in Uruguay. P. RUBINO and V. FUENTES. An. Fac. de med., Univ. de Montevideo, 1926, xi, 357.

Mistaken diagnoses of intestinal parasites. R. H. HARRINGTON. Virginia M. Month., 1926, liii, 579.

Diverticulum ileus. II. Intestinal obstruction resulting from diverticulum formation in the large and small bowel. F. KASPAR. Arch. f. klin. Chir., 1926, cxli, 389.

A case of diverticula of the intestine in an infant of 6 weeks. CRESSON. Bull. et. mém. Soc. d. chirurgiens de Par., 1926, xviii, 587.

Diverticulitis simulating carcinoma. A. W. SHERRILL. Atlantic M. J., 1926, xxx, 165.

What is chronic constipation? Some common fallacies in its diagnosis and management. E. L. GARDNER. Minnesota Med., 1926, ix, 678.

The problem of obstipation and surgery. E. PAYR. Verhandl. d. Gesellsch. f. Verdauungs- u. Stoffwechselkrankh., 1926, p. 141, 180.

The study of mesenterial openings with particular reference to so-called transhaesio intestini. A. CHRIST. Zentralbl. f. allg. Path. u. path. Anat., 1926, xxxviii, 6.

Note on a case of abnormal intestinal rotation. H. B WALKER. Edinburgh M. J., 1926, n.s. xxxiii, 725.

Intestinal obstruction. J. B. DEAVER. Ann. Surg., 1926, lxxxiv, 571. [282]

Intestinal obstruction: a survey of 135 personal cases. W. B. HOLDEN. Arch. Surg., 1926, xiii, 882.

Acute intestinal obstruction. ROCHET. Lyon chir., 1926, xxiii, 651.

Two unusual cases of intestinal obstruction. SANTY and GELAS. Lyon chir., 1926, xxiii, 616.

A case of double intestinal obstruction. L. F. O'SHAUGHNESSY. Lancet, 1926, ccxi, 1163.

A case of postoperative intestinal obstruction. ROUFFART, WEYMEERSCH, NISOT, and MAINJOT. Bull. Soc. d'obst. et de gynéc. de Par., 1926, xv, 546.

Intestinal obstruction by foreign body in the intestinal tract. H. V. OLASCOAGA. An. Fac. de med., Univ. de Montevideo, 1926, xi, 395.

Intestinal obstruction due to a large calcified fibroma. E. WEBER. Bull. Soc. d'obst. et de gynéc. de Par., 1926, xv, 523.

The treatment of acute mechanical occlusion of the intestines. W. KOERTE. Arch. f. Verdauungskr., 1926, xxxvii, 83. [283

The operative treatment of chronic intestinal stasis. A. ZENO. Rev. de cirug., Buenos Aires, 1926, v, 433.

A case of multiple polyposis of the intestine. R. G. ALLEY. Clifton M. Bull., Clifton Springs, N. York, 1926, xii, 141.

Precancerous diseases of the intestine, especially in polyposis. SCHMIEDEN. 50 Tag. d. deutsch. Ges. f. Chir., Berlin, 1926. [284]

Coincident cancer and melanosis of the bowel; with the report of a case. M. F. PORTER. Surg., Gynec. & Obst., 1926, xliii, 744.

Volvulus of the small intestine. PETIT DE LA VILLÉON. Bull. et mém. Soc. d. chirurgiens de Par., 1926, xviii, 583.

Two cases of late postoperative volvulus of the small intestine. COTTE and WERTHEIMER. Lyon chir., 1926, xxiii, 623.

Intussusception, with a report of six cases. G. A. NEUFFER. J. South Carolina M. Ass., 1926, xxii, 250.

Recurrent intussusception. W. B. SWETE-EVANS. J. Roy. Army Med. Corps, Lond., 1926, xlvii, 448.

Ileus, partial and complete. P. B. CHAMPLIN. J. Oklahoma State M. Ass., 1926, xix, 319.

Ileus and dilatation. BRENTANO. Zentralbl. f. Chir., 1926, liii, 2278.

Food substances as causes of ileus. J. LOESSL. Zentralbl. f. Chir., 1926, liii, 2327.

Traumatic retroperitoneal rupture of the duodenum; traumatic intraperitoneal and extraperitoneal rupture of the duodenum; strangulated Meckel's diverticulum in the right femoral canal; solitary non-parasitic cyst of the liver. S. HARRINGTON. Surg. Clin. N. Am., 1926, vi, 1185. [285]

Diverticulum of the duodenum. K. W. KINARD and D. T. VANDEL. J. Missouri State M. Ass., 1926, xxiii, 430.

Duodenal ulcers in children. L. B. DICKEY. Am. J. Dis. Child., 1926, xxxii, 872.

The diagnostic value of thoracic pain in perforating ulcers of the duodenum. E. PČSMAREST. Noticias med., 1926, i, 29.

The medical aspects of duodenal ulcer. H. C. MOFFITT. Canadian M. Ass. J., 1926, xvi, 1044. [285]

The surgical treatment of duodenal ulcer. F. N. G. STARR. Canadian M. Ass. J., 1926, xvi, 1051. [285]

The radiological aspect of duodenal ulcer. W. H. DICKSON. Canadian M. Ass. J., 1926, xvi, 1053. [285]

Occult postoperative duodenal fistula. A. WINKELBAUER. Deutsche Ztschr. f. Chir., 1926, cxcvi, 232.

Peptic ulcer of the jejunum. H. HABERER. Arch. f. klin. Chir., 1926, cxl, 395.

Jejunal ulcer following gastro-enterostomy. C. J. MACGUIRE, JR. Ann. Surg., 1926, lxxxiv, 890.

A large jejunocutaneous fistula. R. FINOCHIETTO. Arch. argent. de enferm. d. apar. digest., 1926, ii, 32.

Retrograde invagination of the ileum due to a gastroenterostomy. A. FIORENTINI. Policlin., Rome, 1926, xxxiii, sez. chir., 456.

Perforation of a Meckel's diverticulum due to the swallowing of a needle. H. KUNZ. Wien. med. Wchnschr., 1926, lxxvi, 436.

An unusual complication of a persistent Meckel's diverticulum. N. L. ECKHOFF. Guy's Hosp. Rep., Lond., 1926, lxxvi, 429.

Carcinoma of the small intestine; report of a case of carcinoma of the ileum. E. D. CLARK. Surg., Gynec. & Obst., 1926, xliii, 757.

The human large intestine in the newborn and in the adult. J. W. LARIMORE. Ann. Clin. Med., 1926, v, 439.

The colon as a urinary receptacle. C. H. MAYO and L. D. POWELL. Surg. Clin. N. Am., 1926, vi, 1131. [287]

The technique for roentgenological study of the colon. W. O. UPSON. Am. J. Roentgenol., 1926, xvi, 419.

A case of megacolon and megasigmoid. SCHEMMEL. Med. Klin., 1926, xxii, 887.

The etiology and treatment of Hirschsprung's disease. K. STEINTHAL. Beitr. z. klin. Chir., 1926, cxxxvii, 401.

Family diverticulosis of the colon. F. W. MACKOY. Radiology, 1926, vii, 498.

A case of total removal of the large intestine for a chronic ulcerating colitis polyposa. MOSZKOWICZ. Zentralbl. f. Chir., 1926, liii, 1696.

Cancer of the large intestine; therapeutic indications. V. PAUCHET. Arch. argent. de enferm. d. apar. digest., 1926, ii, 21.

An unusual case of removal of the colon. S. A. LANE. Lancet, 1926, ccxi, 1163.

Round ulcer of the cæcum. MARWEDEL. Zentralbl. f. Chir., 1926, liii, 2406.

Lymph cysts of the cæcum. FLICK. Med. Klin., 1926, xxii. 1071.

Primary inflammatory tumor of the cæcum without appendicitis. J. A. LAZARUS. Am. J. Surg., 1926, n.s. i, 350.

Traumatic perforation of the normal appendix. R. GUTZEIT. Zentralbl. f. Chir., 1926, liii, 1943.

Axial rotation of the appendices epiploicæ simulating appendicitis. K. W. BENDER. Zentralbl. f. Chir., 1926, liii, 1172.

Torsion of the meso-appendix with gangrene of the appendix. G. D. F. MCFADDEN. Brit. M. J., 1926, ii, 1223.

An unusual anomaly of position of the appendix. H. KOEHLER. Zentralbl. f. Chir., 1926, liii, 1115.

The cause of acute appendicitis—the hydromechanics in acute appendicitis. C. VAN ZWALUWENBURG. California & West. Med., 1926, xxv, 612.

The so-called benign character of appendicitis. SANTY. Lyon chir., 1926, xxiii, 646.

The kinds of appendicitis that yield to physical therapy. R. T. MORRIS. Am. J. Surg., 1926, n.s. i, 357.

Acute appendicitis. H. K. WALLACE. J. Missouri State M. Ass., 1926, xxiii, 434.

Acute appendicitis in childhood. B. B. BLOTZ. Colorado Med., 1926, xxiii, 392.

A case of perforated appendicitis. GIRONÉS. Arch. de med., cirug. y especial., 1926, vii, 35.

The mortality in appendicitis. A. E. ARCHER and M. A. R. YOUNG. Canadian M. Ass. J., 1926, xvi, 1491.

The mortality rate in appendicitis—is it on the increase? A. W. MARSH. Boston M. & S. J., 1926, cxcv, 1059.

Appendicitis in a patient with a fetal type of ileum and cæcum giving rise to gall-bladder symptoms. L. P. GLOVER. J. Am. M. Ass., 1926, lxxxvii, 2092.

A case of ascaridian appendicitis. PERRIN. Lyon chir., 1926, xxiii, 628.

Subhepatic appendicitis. M. TUSSAU. Bull. et mém. Soc. d. chirurgiens de Par., 1926, xviii, 598.

Appendicitis in a case of liver cirrhosis and splenomegaly. T. WILHELM. Zentralbl. f. Chir., 1926, liii, 2392.

Hæmaturia in appendicitis. E. SCIAKY. Policlin., Rome, 1926, xxxiii, sez. prat., 1353.

Carcinoids of the appendix. L. AGUILAR. Bol. Soc. de cirug. de Chile, 1926, iv, 173.

Chronic appendicitis from the standpoint of the surgeon. H. VON HABERER. Verhandl. d. Gesellsch. f. Verdauungs- u. Stoffwechselkrankh., 1926, p. 204, 219.

Why, when, and how should appendicectomy be performed? L. MAYER. Bruxelles-méd., 1926, vi, 1530.

The question of unsuccessful appendectomies. KELLING. 50 Tag. d. deutsch. Ges. f. Chir., Berlin, 1926. [288]

Intestinal obstruction following appendectomy. G. W. CARLSON and V. F. MARSHALL. Ann. Surg., 1926, lxxxiv, 583. [288]

A self-inflicted abdominal wound with injury to the transverse colon and omentum. A. A. BALD. Nebraska State M. J., 1926, xi, 479.

The removal of an artificial anus of the sigmoid flexure. SONNTAG. Zentralbl. f. Chir., 1926, liii, 1089.

Malignant adenoma of the sigmoid. H. A. LEDIARD. Edinburgh M. J., 1926, n.s. xxxiii, 729.

Total colectomy as a treatment of the megasigmoid; end-to-side anastomosis. P. L. MIRIZZI. Arch. Surg., 1926, xiii, 837.

Megalocolon of the descending portion of the large intestine in a patient subjected four years previously to resection of the sigmoid for volvulus. H. GROVE, C. SORROLLA, A. SCHOENHER, and COSTA. Bol. Soc. de cirug. de Chile, 1926, iv, 153. Arch. Soc. méd. de Valparaiso, 1926, i, 77.

Rectoscopy and sigmoidoscopy. R. BENSAUDE. 1926: Paris, Masson.

Rectoscopy in atonic obstipation and its operative treatment. V. SCHMIEDEN. Med. Klin, 1926, xxii, 1364.

Clinical variations in negro proctology: the venereal factor. C. ROSSER. J. Am. M. Ass., 1926, lxxxvii, 2084.

The treatment of strictures of the rectum with diathermy. H. PICARD. Ztschr. f. d. ges. phys. Therap., 1926, xxxii, 45.

The internal treatment of chronic rectal hæmorrhages. W. GOLDSCHMIDT. Wien. med. Wchnschr., 1926, lxxvi, 1086.

A rectal biopsy punch. J. F. MONTAGUE. Am. J. Surg., 1926, n.s. i, 375.

Carcinoma of the rectum in the young. S. R. SCHULTZ. Med. Times, 1926, liv, 297.

Sacral anæsthesia in operations on the rectum. J. S. LUNDY and C. F. MCCUSKEY. Atlantic M. J., 1926, xxx, 14. [288]

Imperforate anus. J. GARRIDO-LESTACHE. Pediat. espan., 1926, xv, 273.

Pruritus ani and pruritus vulvæ of mycotic origin. A. CASTELLANI. Practitioner, 1926, cxvii, 341.

The causal therapy of hæmorrhoidal disease. GRONAU. Deutsche med. Wchnschr., 1926, lii, 1558.

The principles and technique of the surgical treatment of hæmorrhoids. R. W. GILES. J. Nat. M. Ass., 1926, xviii, 202.

The treatment of extrasphincteric anal fistulæ. DANIELS. Zentralbl. f. Chir., 1926, liii, 2469.

A case of perineal excision of anorectal cancer. S. C. D. GUPTA. Indian M. Gaz., 1926, lxi, 602.

Liver, Gall Bladder, Pancreas, and Spleen

The measurement of the liver by means of roentgen rays, based upon a study of 502 subjects. G. E. PFAHLER. Am. J. Roentgenol., 1926, xvi, 558.

Changes in basculation and anteroversion of the liver in right-sided and symmetrical splanchnoptosis. T. MARTINI. Arch. argent. de enferm. d. apar. digest., 1926, ii, 63.

Handbook of biological methods: E. ABDERHALDEN. Vol. IV. Related chemical and physical methods. Part VI, Section 2, No. 1. A study of the function of the digestive apparatus. Functional tests of the liver by means of injection of substances into the portal circulation: E. REISS. Chemical functional test of the liver in man: E. REISS and G. LEPEHNE. Newer methods of testing liver function: G. LEPEHNE. The development of Eck's fistula in the dog. Liver extirpation in the dog. Extirpation of the liver in the goose: F. FISCHLER. 1926: Berlin, Urban & Schwarzenberg.

Functional liver tests in massive hepatic trauma. L. A. LEVISON and E. J. MCCORMICK. Ann. Clin. Med., 1926, v, 483.

The surgical significance of experimental studies concerning the sites of formation of bile pigment. J. L. BOLLMAN, C. SHEARD and F. C. MANN. Surg. Clin. N. Am., 1926, vi, 1257. [289]

The action of calomel on the bile. W. F. VON OETTINGEN and T. SOLLMANN. J. Am. M. Ass., 1926, lxxxvii, 1990.

Experimental and clinical investigations in cholæmia. WILDEGANS. 50 Tag. d. deutsch. Ges. f. Chir., Berlin, 1926. [289]

Physiological considerations in the treatment of obstructive jaundice. W. WALTERS. J. Am. M. Ass., 1926, lxxxvii, 2153.

Surgical treatment in cases of obstructive jaundice. E. S. JUDD and D. B. COBB. Practitioner, 1926, cxvii, 356.

The operative findings in a case of jaundice. E. M. DALAND. Boston M. & S. J., 1926, cxcv, 1113.

Leptospirosis icterohæmorrhagica (Weil's disease), with the report of a fatal case. J. E. BATES. Canadian M. Ass. J., 1926, xvi, 1466.

A case of hydatid disease of the liver with obstruction of the common bile duct by a daughter cyst. F. C. BURKE-GAFFNEY. Med. J. Australia, 1926, ii, 804.

Hydatid cyst of the liver simulating a calculus implanted in the common duct. J. ARNELLO. Bol. Soc. de cirug. de Chile, 1926, iv, 157.

Tumorous hepatic tuberculosis. F. E. RUFZ and L. GONZÁLEZ SABATHIE. Rev. Soc. argent. de biol., 1926, ii, 508.

Tertiary syphilis of the liver. T. MCCRAE and W. R. CAVEN. Am. J. M. Sc., 1926, clxxii, 781.

The roentgenological diagnosis of liver abscess with or without subdiaphragmatic abscess. H. K. PANCOAST. Am. J. Roentgenol., 1926, xvi, 303. [290]

Intrahepatic lithiasis associated with multiple internal biliary fistulæ. A. H. MCINDOE. Surg. Clin. N. Am., 1926, vi, 1233. [290]

Drainage of the bile passages by Wischnewsky's method. A. GUTIERREZ. Rev. de cirug., Buenos Aires, 1926, v, 476.

Some points bearing on the surgery of the biliary tract. J. W. G. GRANT. Practitioner, 1926, cxvii, 376.

Contributions on the surgery of the bile passages. PETERMANN. Zentralbl. f. Chir., 1926, liii, 2471.

Temporary clamping off of the hepatoduodenal ligament for bloodless operations on the liver. S. I. DUCHINOVA. Vestnik chir. i pagranicnych oblastej, 1925, v, 34. [290]

Gall-bladder problems. H. M. RICHTER. Med. Herald & Physiotherap., 1926, xlv, 315.

Consideration of the gall bladder with reference to the process of emptying. G. M. HIGGINS and F. C. MANN. Surg. Clin. N. Am., 1926, vi, 1241. [291]

A contribution on the motility of the gall bladder. D. CAMPANACCI and M. GROPPALI. Klin. Wchnschr., 1926, v, 1639.

Volvulus of the gall bladder with gangrene. JENCKEL. Deutsche Ztschr. f. Chir., 1926, cxcvii, 171.

Cholecystography. K. HERMANN. Brit. J. Radiol., 1926, xxxi, 491.

Cholecystography. W. H. COLE. J. Kansas M. Soc., 1926, xxvi, 386.

Cholecystography. W. M. SHERIDAN. J. South Carolina M. Ass., 1927, xxiii, 266.

The present status of cholecystography. B. R. MOONEY. Canadian M. Ass. J., 1926, xvi, 1461.

Roentgenological diagnosis of gall-bladder disease. J. ROEMER. J. Med. Soc. N. Jersey, 1926, xxiii, 597.

Regarding the roentgenological representation of the pathologically altered (enlarged) gall bladder. M. L. BISCHOFF. Brit. J. Radiol., 1926, xxxi, 506.

Further advancements in the technique and interpretation of cholecystography by the oral method. W. H. STEWART and E. J. RYAN. N. York State J. M., 1926, xxvi, 819. [291]

Non-secretion of tetra-iodophenolphthalein. A. S. KIRKLAND. Radiology, 1926, vii, 512.

Some new phases of the physiology of the biliary tract. E. A. GRAHAM, W. H. COLE, G. H. COPHER, and S. KODAMA. Ann. Surg., 1926, lxxxiv, 343. [291]

Surgical aspects of certain phases of liver function. E. P. RICHARDSON. Ann. Surg., 1926, lxxxiv, 352. [291]

Problems in gall-bladder surgery. E. R. MCGUIRE. Ann. Surg., 1926, lxxxiv, 366. [291]

Dangers incident to cholecystectomy. H. W. CAVE. Ann. Surg., 1926, lxxxiv, 371. [291]

Surgical management of the complications of cholecystitis. J. B. DEAVER and V. G. BURDEN. Ann. Surg., 1926, lxxxiv, 379. [291]

Association of cholecystitis with duodenal ulcer. H. A. BRUCE. Ann. Surg., 1926, lxxxiv, 387. [291]

Strictures and operative injuries of the bile ducts. J. DOUGLAS. Ann. Surg., 1926, lxxxiv, 392. [291]

The operative management of common duct stones. G. W. CRILE. Ann. Surg., 1926, lxxxiv, 411. [291]

Functions and diseases of the gall bladder: the value of cholecystography in diagnosis. E. A. GRAHAM. Brit. M. J., 1926, ii, 671.

The diagnosis and treatment of cholecystitis. A. F. HURST. Brit. M. J., 1926, ii, 676.

An analysis of 482 gall-bladder cases. J. M. HITZROT and N. W. CORNELL. Ann. Surg., 1926, lxxxiv, 829.

Gall-bladder disease: some observations on a series of 135 cases. A. J. BLAXLAND. Practitioner, 1926, cxvii, 365.

The diagnosis of cholecystic disease. H. W. BACHMAN. Virginia M. Month., 1926, liii, 576.

The etiology and pathology of diseases of the gall bladder. R. A. MORISON. Virginia M. Month., 1926, liii, 572.

Tænia saginata in the gall bladder. E. B. BENEDICT. J. Am. M. Ass., 1926, lxxxvii, 1917.

The biology of gall stones. W. SCHOSSERER. Wien. med. Wchnschr., 1926, lxxvi, 290, 372.

A clinical type of cholelithiasis resembling renal disease; report of two cases. R. S. CORBETT and C. B. PEIRCE. Surg., Gynec. & Obst., 1926, xliii, 459. [294]

The stoneless gall bladder: a study of postoperative end-results. E. M. STANTON. J. Am. M. Ass., 1926, lxxxvii, 2160.

Duodenal treatment of diseases of the gall bladder and the bile passages. E. ALLARD. Zentralbl. f. innere med., 1926, xlvii, 336.

Should the gall bladder be removed without drainage? O. H. WANGENSTEEN. Ann. Surg., 1926, lxxiv, 821.

Complications incident to the operative treatment of inflammatory diseases of the gall bladder. A. P. HEINECK. Virginia M. Month., 1926, liii, 603.

The question of faulty ligature of the cystic duct in cholecystectomy. C. FLECHTENMACHER. Zentralbl. f. Chir., 1926, liii, 1625.

A clinical study of the results of 470 operations on the gall bladder. T. B. JOHNSON and A. A. PEARRE. South. M. J., 1926, xix, 889.

Methods of diagnosing diseases of the pancreas; Wohlgemuth's reaction. S. AVENDAÑO. Arch. Soc. méd. de Valparaíso, 1926, i, 66.

Acute pancreatitis. R. J. MCN. LOVE. Lancet, 1926, ccxi, 1262.

Acute hæmorrhagic gangrenous pancreatitis. K. S. J. HOHLEN. Nebraska State M. J., 1926, xi, 477.

The early diagnosis of acute pancreatic necrosis. E. UNGER and H. HEUSS. Zentralbl. f. Chir., 1926, liii, 1500.

Pancreatic tumors, malignant and benign. M. EINHORN. Am. J. M. Sc., 1926, clxxii, 796.

True cysts of the spleen. O. MONDRÉ. Zentralbl. f. Gynaek., 1926, l, 2111.

Unusual diseases of the spleen and postoperative complications after splenectomy. W. DOBRZANIECKI. Zentralbl. f. Chir., 1926, liii, 1940.

A case of wandering spleen—splenectomy. J. B. VAIDYA. Indian M. Gaz., 1926, lxi, 601.

Ruptured spleen: splenectomy; recovery. I. R. ROSIN. Brit. M. J., 1926, ii, 1049.

Investigation of a case of Henoch's purpura treated by splenectomy. B. MYERS, A. K. GORDON, and R. MAINGOT. Brit. J. Child. Dis., 1926, xxiii, 241.

Indications and results of splenic operations in hepatosplenic diseases. E. RANZI. Verhandl. d. Gesellsch. f. Verdauungs-u. Stoffwechselkrankh., 1926, p. 289.

Miscellaneous

Grave traumatism of the left hypochondrium with partial laceration of the renal artery. A. BERHOUET. An. Fac. de med., Univ. de Montevideo, 1926, xi, 387.

Three cases of wounds of the hypochondrium from revolver bullets with multiple visceral lesions. P. MOIROUD and J. L. ROUX-BERGER. Bull. et mém. Soc. nat. de chir., 1926, lii, 863.

Foreign bodies in the abdomen. L. SCHMIDT. Zentralbl. f. Gynaek., 1926, l, 2563.

Abdominal tuberculosis. C. S. COLVIN. Med. J. Australia, 1926, ii, 840.

Diabetic coma and an acute abdomen. L. S. MCKITTRICK. Boston M. & S. J., 1926, cxcv, 1113.

Stercoraceous subphrenic abscess following necrotic appendicitis. O. CANTELMO. Rassegna internaz. di clin. e terap., 1926, vii, 605.

Amœbiasis and abdominal granulomatosis. A. E. BIANCHI. Bol. inst. de clín. quir., 1926, ii, 289.

Alveolar echinococcus of the right liver and kidney region. K. HELLY. Zentralbl. f. allg. Path. u. path. Anat., 1926, xxxvii, 310.

The clinical diagnosis of abdominal tumors. T. NAEGELI. 1926: Munich, Bergmann.

The gall bladder in relation to right-sided pain. MCL. ROGERS. J. Oklahoma State M. Ass., 1926, xix, 315.

Right abdominal pain—"kidney." R. M. HOWARD. J. Oklahoma State M. Ass., 1926, xix, 317.

Right side pain and its relation to appendicitis. W. H LIVERMORE. J. Oklahoma State M. Ass., 1926, xix, 318.

Right abdominal pain of thoracic origin. L. J. STARRY. J. Oklahoma State M. Ass., 1926, xix, 323.

Central nervous disease and pain in the right side of the abdomen. A. D. YOUNG. J. Oklahoma State M. Ass., 1926, xix, 324.

The essentials and treatment of colicky pain. J. PAL. Am. Med., 1926, xxxii, 774.

Ultraviolet radiation in cœliac disease. R. G. MICHELMORE. Lancet, 1926, ccxi, 1264.

Splanchnic anæsthesia in the surgery of the upper abdomen. O. TOENNIESSEN. Ergebn. d. chir. u. Orthop., 1926, xix, 683.

Abdominal incisions: an attempt at standardization. H. PEET. Med. J. Australia, 1926, ii, 790.

The indications for the technique of drainage in the presence of abdominal inflammation. HANS. Zentralbl. f. Chir., 1926, liii, 2413.

A new sign in the examination of diaphragmatic function. F. PARODI. Policlin., Rome, 1926, xxxiii, sez. prat., 1320.

GYNECOLOGY

Uterus

The action of the sympathetic upon the blood circulation of the uterus. L. MICHON and H. QUINCIEU. Lyon chir., 1926, xxiii, 549.

The iron metabolism in the uterus. G. MOTTA. Arch. di ostet. e ginec., 1926, 2 s. xiii, 348.

The treatment of uterine retrodeviations. V. MARZETTI. Gynéc. et obst., 1926, xiv, 233.

Ligamentopexy with interposition of the uterus in the treatment of certain forms of prolapse. COTTE. Lyon chir., 1926, xxiii, 641.

Acute inversion of the uterus. J. C. MASSON. Surg. Clin. N. Am., 1926, vi, 1329. [295]

Hysterectomy by the vaginal route for complete prolapse in the octogenarian nullipara. SCHOCKAERT, HENROTAY, and BROUHA. Bull. Soc. d'obst. et de gynéc. de Par., 1926, xv, 546.

Hæmostasis in vaginal hysterectomy for procidentia. A. B. SPALDING. Am. J. Obst. & Gynec., 1926, xii, 655.

The fibril content of the uterine mucous membrane in clinically diagnosed genital hypofunction. F. WERMBTER. Zentralbl. f. Gynaek., 1926, l, 1626.

A case of unusual uterine malformation. J. PÉRY. Bull. Soc. d'obst. et de gynéc. de Par., 1926, xv, 491.

The uterine cervix in the pathogenesis of sterility. E. A. FOX. Bol. Soc. de obst. y ginec. de Buenos Aires, 1926, v, 335.

Uterography, its technique and value. H. NAHMMACHER. Zentralbl. f. Gynaek., 1926, l, 2238.

Hæmorrhage from the non-gravid uterus following trauma. D. M. LINDSAY. J. Obst. & Gynec. Brit. Emp., 1926, xxxiii, 648.

Curettage of the uterus, its indications and advantages. H. E. MILLER. Am. J. Obst. & Gynec., 1926, xii, 860.

Ovarian hyperplasia of the uterine mucous membrane. A. BABES. Zentralbl. f. Gynaek., 1926, l, 2639.

The histological study of chronic endometritis. A. SCARPELLO. Arch. di ostet. e ginec., 1926, 2 s. xiii, 363.

Functional uterine hæmorrhage. G. W. WATERMAN. Rhode Island M. J., 1926, ix, 189.

The radium treatment of benign uterine hæmorrhage. D. P. MURPHY. South M. & S., 1926, lxxxviii, 797.

The causes and treatment of uterine hæmorrhage. B. WHITEHOUSE and W. F. SHAW. Brit. M. J., 1926, ii, 723. [295]

Calcification of the uterus. M. ESSER. Monatsschr. f. Geburtsh. u. Gynaek., 1926, lxxiv, 265.

Cholesterinæmia and tumors of the uterus. E. DEBELLA. Arch. di ostet. e ginec., 1926, 2 s. xiii, 337.

The clinical significance of mucous polyps of the uterus. O. BUETTNER. Schweiz. med. Wchnschr., 1926, lvi, 718.

Cervical fibroma. BOTTARO. Rev. méd. d. Uruguay, 1926, xxix, 222.

Pediculated subserous fibroma presenting at its surface four large cysts. R. SCHOCKAERT. Bruxelles-méd., 1926, vi, 1566.

Evolution of new fibromata in the uterus with a calcified fibroma. G. JEANNENEY. Bull. Soc. d'obst. et de gynéc. de Par., 1926, xv, 484.

Sarcomatous degeneration of uterine fibromyomata with multiple metastases. E. GREENE. Bol. Soc. de cirug. de Chile, 1926, iv, 181.

Myomectomy, hysterectomy, and radiotherapy in fibromyoma of the uterus. J. C. MASSON. J. Am. M. Ass., 1926, lxxxvii, 1530.

A contribution on lipomata of the uterus. H. DWORZAK. Frankfurt. Ztschr. f. Path., 1926, xxxiv, 20.

Cystic cervical lymphangioma. JOB. Bull. Soc. d'obst. et de gynéc. de Par., 1926, xv, 514.

Heterotopic mixed tumor of the corpus uteri. G. HALTER. Zentralbl. f. Gynaek., 1926, l, 2194.

Precancerous conditions of the uterus. C. A. CASTAÑO. Bol. Soc. de obst. y ginec. de Buenos Aires, 1926, v, 369.

The question of carcinoma of the stump after supravaginal amputation of the myomatous uterus. F. ISBRUCH. Zentralbl. f. Gynaek., 1926, l, 1771.

An apparatus for measuring and applying roentgen radiation in carcinoma of the uterine cervix. T. BRANDESS. Strahlentherapie, 1926, xxii, 569.

The operability of inoperable uterine carcinoma after radiation. H. SEIDEMANN. Strahlentherapie, 1926, xxii, 554.

The relation between the treatment of cancer of the cervix and the cell type. F. A. PEMBERTON. Am. J. Obst. & Gynec., 1926, xii, 536. [295]

The prognosis of cervical carcinoma as determined by biopsy. B. LIEGNER. Zentralbl. f. Gynaek., 1926, l, 2485.

A comparative review of the results of various methods of treating uterine cancer. T. N. ILJIN. Zentralbl. f. Gynaek., 1926, l, 2567.

Hydatidiform mole, with a case report. E. N. BEATTY. Cincinnati J. M., 1926, vii, 560.

Adnexal and Periuterine Conditions

The interstitial gland of the ovary. V. CONILL. Ars. med., 1926, ii, 238.

The ovarian hormone. M. HARTMANN and H. ISLER. Biochem. Ztschr., 1926, clxxv, 46

The ovarian hormone and its clinical use. B. ZONDEK. Klin. Wchnschr., 1926, v, 1218.

Concerning female sexual hormones. S. LOEWE and E. H. V. VOSS. Klin. Wchnschr,. 1926, v, 1083.

The influence of the corpus luteum upon menstruation. B. WHITEHOUSE. J. Obst. & Gynec. Brit. Emp., 1926, xxxiii, 380. [296]

The specific function of the ovary in the female organism and the organotherapeutic use of ovarian preparations. A. HEYN. Deutsche med. Wchnschr., 1926, lii, 1333.

Ovarian preparations and anaphylaxis. P. BRAUN. Fortschr. d. Med., 1926, xliv, 910.

Biological and therapeutic studies with ovarnon, a new dry ovarian preparation. A. HEYN. Med. Klin., 1926, xxii, 1457.

The possibility of influencing suitable cases of primary sterility by operative procedures on the ovary and the tubal opening. F. ISBRUCH. Zentralbl. f. Gynaek., 1926, l, 1705.

Loss of ovarian function after roentgen castration. R. CORDUA. Zentralbl. f. Gynaek., 1926, l, 2354.

Further observations on "roentgen-children." P. WERNER. Arch. f. Gynaek., 1926, cxxix, 157.

The present status of our knowledge of the effect of irradiation upon the generative organs and the offspring. H. J. BAGG. Am. J. Roentgenol., 1926, xvi, 529.

Calcified ovary: considerations on sterility. HENROTAY and KEIFFER. Bull. Soc. d'obst. et de gynéc. de Par., 1926, xv, 545.

Dental formations in an ovarian cyst. PONT. Lyon chir., 1926, xxiii, 610.

Appendicular ovarian lesions. L. DIEULAFÉ. Bull. Soc. d'obst. et de gynéc. de Par., 1926, xv, 542.

Suppurative degeneration of the ovary of subacute evolution simulating pregnancy. A. DESCAMPS. Bull. Soc. d'obst. et de gynéc. de Par., 1926, xv, 522.

The histological changes in ovarian transplants in animals. R. CALVANICO. Rassegna internaz. di clin. e terap., 1926, vii, 585.

Further observations on metaplastic bone formation in ovarian transplants. H. E. V. VOSS. Biol. gen., 1926, ii, 1.

Utero-ovarian pararenal and renal tumors: differential diagnostic considerations. C. VERCESI. Clin. ostet., 1926, xxviii, 465.

An analysis of 500 tumors of the ovary. G. L. ROHDENBURG. J. Lab. & Clin. Med., 1926, xii, 211.

The régime of development of cysts of the ovary. TÉDENAT. Bull. Soc. d'obst. et de gynéc. de Par., 1926, xv, 503.

An unusually large ovarian cyst. J. H. GIRVIN. Am. J. Obst. & Gynec., 1926, xii, 896.

Fibroma of the ovary. DELFOURD and LUCIEN. Bull. Soc. d'obst. et de gynéc. de Par., 1926, xv, 512.

Pure fibroma of the ovary. F. PATTI. Arch. di ostet. e ginec., 1926, 2 s. xiii, 273.

Giant tumor of the ovary. DEROUVILLE, MADON, and VILLA. Bull. Soc. d'obst. et de gynéc. de Par., 1926, xv, 496.

Endometrioma of the ovary. A. L. MCILROY. Proc. Roy. Soc. Med., Lond., 1926, xx, 117.

The origin of malignant tumors of the ovary. A. J. BENGOLEA and A. J. PAVLOVSKY. Bol. Soc. de obst. y ginec. de Buenos Aires, 1926, v, 348.

Bilateral epitheliomatous tumor of the ovary with ascites. BÉGOUIN and MASSÉ. Bull. Soc. d'obst. et de gynéc. de Par., 1926, xv, 495.

Primary carcinoma of the ovary. B. MANN. Am. J. Obst. & Gynec., 1926, xii, 894.

Spindle-celled sarcoma of the ovary with a twisted pedicle. L. S. SCHEFFEY. Am. J. Obst. & Gynec., 1926, xii, 896.

Experimental investigations on tubal movements. F. VON MIKULICZ-RADECKI. Arch. f. Gynaek., 1926, cxxviii, 318.

Cyclic changes in the spontaneous contractions of the human fallopian tube. D. L. SECKINGER and F. F. SNYDER. Bull. Johns Hopkins Hosp., Balt., 1926, xxxix, 371.

Roentgenography of the cavity of the uterus and fallopian tubes, with special reference to its value in cases of sterility. R. L. MCCREADY and E. J. RYAN. Am. J. Roentgenol., 1926, xvi, 321. [297]

Some aspects of tubal inflation. L. M. RANDALL. Med. Clin. N. Am., 1926, x, 689.

Irradiation in disease of the abdominal opening of the fallopian tube or in uterus masculinus. M. N. LAPINSKY. Monatsschr. f. Psychiat u. Neurol., 1926, lxi, 248.

Normal delivery after implantation of the tubes. F. UNTERBERGER. Monatsschr. f. Geburtsh. u. Gynaek., 1926, lxxiii, 1.

Large hydrosalpinx. DELFOURD and LUCIEN. Bull. Soc. d'obst. et de gynéc. de Par., 1926, xv, 516.

Hydrosalpinx with bacillus coli infection. M. J. GELPI. Am. J. Obst. & Gynec., 1926, xii, 901.

Report of 100 consecutive cases of salpingitis. G. H. REESE. Virginia M. Month., 1926, liii, 589.

Suppurative salpingitis opening freely into the pelvic cavity. BAILLAT and MOREL. Bull. Soc. d'obst. et de gynéc. de Par., 1926, xv, 543.

Double pyosalpinx followed by immediate pregnancy. M. RIVIÈRE. Bull. Soc. d'obst. et de gynéc. de Par., 1926, xv, 494.

Intervention for bilateral pyosalpinx with fistulæ of the rectum, bladder, and skin. GUYOT, PRINCETEAU, and MAILLE. Bull. Soc. d'obst. et de gynéc. de Par., 1926, xv, 490.

A case of adnexal tuberculosis. GUYOT and PRINCETEAU. Bull. Soc. d'obst. et de gynéc. de Par., 1926, xv, 485.

Intra-abdominal hæmorrhage due to spontaneous rupture of the non-gravid tube: a contribution on the genesis of intra-abdominal hæmorrhages arising from the female genitalia. R. KOEHLER. Arch. f. klin. Chir., 1926, cxl, 628.

Conservative treatment in inflammatory lesions of the adnexa. M. CAZIN. Paris chir., 1926, xviii, 209.

External Genitalia

The treatment of granuloma inguinale of the vulva with tartar emetic. J. A. MCGLINN. Am. J. Obst. & Gynec., 1926, xii, 665.

Further research in the problem of vulvovaginitis in children. II. Serological studies. E. M. SHERMAN and S. L. NORTON. J. Urol., 1926, xvi, 279. [297]

The treatment of gonorrhœal vulvovaginitis in infants by means of vaccines. L. V. BLANCO and N. M. VILLAZON. Am. J. Dis. Child., 1926, xxxii, 805.

Primary carcinoma of the hymen. O. GERICH. Zentralbl. f. Gynaek., 1926, l, 1372.

Pronounced vaginal atresia of obstetrical origin; consecutive pregnancies; finally complete genital prolapse. LAURENTIE. Bull. Soc. d'obst. et de gynéc. de Par., 1926, xv, 535.

Fibroma of the vagina. TIXIER. Lyon chir., 1926, xxiii, 590.

An unusual case of vaginal tumor. E. VON HOOR. Med. Klin., 1926, xxii, 1112.

Cancer of the entire vagina removed by vulvocolpohysterectomy. CHARBONNEL. Bull. Soc. d'obst. et de gynéc. de Par., 1926, xv, 488.

Miscellaneous

A textbook of gynecology for students and physicians. R. SCHROEDER. 1926: Leipzig, Vogel.

Some important differences in gynecological practice. A. DONALD. Proc. Roy. Soc. Med., Lond., 1926, xx, 117.

Vicious cycles in gynecology. M. G. ROCA. Ars med., 1926, ii, 236.

The static modalities in gynecology. M. L. H. A. SNOW. Med. J. & Rec., 1926, cxxiv, 779.

The material and methods of a gynecological and obstetrical clinic. W. BLAIR BELL. J. Obst. & Gynaec. Brit. Emp., 1926, xxxiii, 642.

The everyday practice of gynecology. O. WILLE. 1926: Leipzig, Kabitzsch.

The study of constitutional types of women. IV. The athletic and sub-athletic constitutional type. J. S. GALANT. Arch. f. Frauenk u. Konstitutions-forsch., 1926, xii, 388.

Periodicity of sex desire. Part I. Unmarried women, college graduates. K. B. DAVIS. Am. J. Obst. & Gynec., 1926, xii, 824.

The phases of the sexual cycle. B. P. WIESNER. Biol. gen., 1926, ii, 137.

Two cases of precocious puberty. G. F. KEATINGE. J. Obst. & Gynæc. Brit. Emp., 1926, xxxiii, 653.

Contributions upon the functional disturbances of puberty. A. VON FEKETE. Arch. f. Gynaek., 1926, cxxviii, 540.

The relation of menstruation to the permeability of the skin capillaries and the autonomic tonus of the skin vessels. W. F. PETERSEN and G. MILLES. Arch. Int. Med., 1926, xxxviii, 730.

The effect of the monthly cycle upon the colloid stability of the plasma. H. EUFINGER. Monatsschr. f. Geburtsh. u. Gynaek., 1926, lxxiv, 139.

The relation between the blood platelets and menstruation. S. Di Francesco. Arch. de ostet. e ginec., 1926, 2 s. xiii, 289.

Dismenol for the relief of menstrual and bladder pains. W. Hofmann. Klin. Wchnschr., 1926, v, 1035.

The treatment of climacteric pains. O. Wieden. Deutsche med. Wchnschr., 1926, lii 796.

Eunuchoid growths of fat in women. J. S. Galant. Anat. Anz., 1926, lxi, 145.

The treatment of female sterility. E. Pouget. Noticias med., 1926, i, 29.

Sterility as a result of unnatural habits of life: an investigation based on animal experimentation and anatomical studies. H. Stieve. 1926: Munich, Bergmann.

The importance of a gynecological examination. Q. U. Newell. J. Missouri State M. Ass., 1926, xxiii, 435.

A new vaginal speculum. S. von Wachenfeldt, Zentralbl. f. Gynaek., 1926, l, 2569.

Headache from the gynecological standpoint. H. S. Crossen. J. Missouri State M. Ass., 1926, xxiii, 429.

The frequency and meaning of backache in gynecology. F. W. Lynch. Am. J. Obst. & Gynec., 1926, xii, 719.

Obscure lesions in gynecology: sensory motor disturbances, painful spasms, and hypotony. P. Petit-Dutaillis. Gynécologie, 1926, xxv, 577.

Ligation of pelvic veins in thrombophlebitis. R. R. Huggins. Am. J. Obst. & Gynec., 1926, xii, 562. **[298]**

Studies on the cytology of the peritoneal fluid in gynecological diseases. E. Vogt. Arch. f. Gynaek., 1926, cxxviii, 363.

Generalities upon inflammation of the pouch of Douglas. R. Condamin. Gynécologie, 1926, xxv, 599.

The uterus and vagina as a source of leucorrhœa; a clinical, chemical, and bacteriological study. R. Schroeder, R. Hinrichs, and R. Kessler. Arch. f. Gynaek., 1926, cxxviii, 94.

The treatment of leucorrhœa. N. Cukor. Med. Klin., 1926, xxii, 1267.

Leucorrhœa and its treatment (modern biological methods). R. Salomon. Westdeutsche Aerztezeit., 1926, xvii.

Protein therapy in gynecology. O. Pankow. Klin. Wchnschr., 1926, v, 1773.

The treatment of leucorrhœa and inflammatory diseases of the adnexa with oriol. W. Schauenstein. Wien. klin. Wchnschr., 1926, xxxix, 922.

The treatment of inflammatory diseases of the lower abdomen with yatrencasein. E. Voelcker. Zentralbl. f. Gynaek., 1926, l, 1777.

The effect of intramuscular injections of milk upon acute inflammatory processes. W. Butomo. Arch. f. Gynaek., 1926, cxxix, 171.

Indications for treatment in inflammatory diseases of the genitalia and peritonitis. W. Benthin. Beihefte z. Med. Klin., 1926, xxii, 75.

Genital tuberculosis in abdominal tuberculosis of the female. O. Gragert. Beitr. z. Klin. d. Tuberk., 1926, lxiii, 768.

A textbook of gonorrhœa: A. Buschke and E. Langer. 1926: Berlin, Springer.

Genital syphilis. Laurentie and Topous-Khay. Bull. Soc. d'obst. et de gynéc. de Par., 1926, xv, 537.

The bleeding time in gynecological diseases. H. Bock. Zentralbl. f. Gynaek., 1926, l, 1387.

Calcium therapy in gynecological practice, with particular reference to hæmosistan. W. Braun. Monatsschr. f. Geburtsh. u. Gynaek., 1926, lxxiv, 160.

Cystocele—with a report of a case of primary cystocele operation. J. L. Bubis. Urol. & Cutan. Rev., 1926, xxx, 705.

Autografts and homografts. De Rouville and Madon. Bull. Soc. d'obst. et de gynéc. de Par., 1926, xv, 507.

A contribution on endometrioid growths. W. Neuweiler. Schweiz. med. Wchnschr., 1926, lvi, 545.

A case of endometrial tumor in a laparotomy scar. Berkeley. J. Obst. & Gynæc. Brit. Emp., 1926, xxxiii, 657.

The question of endometrioid heterotopic epithelial growths and particularly concerning the development of so-called extraperitoneal growths of this type. A. Lauche. Arch. f. klin. Chir., 1926, cxxxix, 785.

Endometriosis of the sac of a right inguinal hernia associated with a pelvic peritoneal endometriosis and an endometrial cyst of the ovary. J. A. Sampson. Am. J. Obst. & Gynec., 1926, xii, 459. **[298]**

The significance of uterine mucosa in the fallopian tube, with a discussion of the origin of aberrant endometrium. E. Novak. Am. J. Obst. & Gynec., 1926, xii, 484. **[298]**

A contribution to the study of endometriosis. C. H. Davis and R. S. Cron. Am. J. Obst. & Gynec., 1926, xii, 526. **[298]**

A case in which endometrial tissue was accidentally implanted. V. Bonney. J. Obst. & Gynæc. Brit. Emp., 1926, xxxxiii, 658.

A large malignant myxoma of the pelvis with thoracici metastasis. Jeanneney and Peyralbe. J. de méd. de Bordeaux, 1926, ciii, 740.

Transabdominal treatment of inoperable cancer. F. Delporte and J. Cahen. Cancer, 1926, iii, 21.

The question of postoperative emphysema of the abdominal wall following gynecological operations. W. Zoepgen. Monatsschr. f. Geburtsh. u. Gynaek., 1926, lxxiii, 201.

OBSTETRICS

Pregnancy and Its Complications

A comparative study of pregnancy and labor in primiparæ in relation to age. L. E. McCaffrey. J. Michigan State M. Soc., 1926, xxv, 649.

The dangers and the duties of the hour in pregnancy. A. E. Hoff. Nebraska State M. J., 1926, xi, 474.

The diagnosis of pregnancy in the first half by fetal roentgenography. A. P. Ramos, S. E. Bermann, and J. A. Saralegui. Semana méd., 1926, xxxiii, 937.

The refractometric method in Abderhalden's reaction. J. Schnupp. Deutsche med. Wchnschr., 1926, lii, 913.

Kermauner's reflex in pregnancy. J. S. Galant. Zentralbl. f. Gynaek., 1926, l, 1663.

Examination of 100 sera tested for Dienst's reaction of pregnancy. K. Fink. Zentralbl. f. Gynaek., 1926, l, 1681.

The ninhydrin reaction as a test of pregnancy. K. von Oettingen. Zentralbl. f. Gynaek., 1926, l, 2555.

Results with the Luettge-von Mertz alcohol-extract reaction. H. Kamniker. Zentralbl. f. Gynaek., 1926, l, 2301.

A critical study of the length of pregnancy in the human being. A Seitz. Klin. Wchnschr., 1926, v, 1665.

Biology and pathology of the female: a handbook of gynecology and obstetrics: J. Halban and L. Seitz. Vol. VII, Part 1. Abnormal duration of pregnancy: L. Nuernberger. Missed labor and premature labor: I. Anatomical Section: L. Nuernberger. II. Clinical Section: T. Heynemann. 1926: Berlin, Urban & Schwarzenberg.

The clinical importance of prolonged pregnancy. J. FRIGYESI. Zentralbl. f. Gynaek., 1926, l, 2253.

On the gastric juice during pregnancy. F. ARZT. Am. J. Obst. & Gynec., 1926, xii, 879.

The influence of the humoral changes in pregnancy upon the red blood picture. G. H. SCHNEIDER. Monatsschr. f. Geburtsh. u. Gynaek., 1926, lxxiv, 14.

The lactic acid content of the blood of pregnant women. G. K. F. SCHULTZE. Zentralbl. f. Gynaek., 1926, l, 1759.

The relation between pregnancy and metabolism. O. L. E. DE RAADT. Ztschr. f. Gynaek. u. Geburtsh., 1926, xc, 63.

The relations between fetal and maternal blood sugar. K. HELLMUTH. Zentralbl. f. Gynaek., 1926, l, 1952.

Studies of the sugar content in the blood and cerebrospinal fluid during pregnancy and in the presence of myomata. K. HELLMUTH. Ztschr. f. Geburtsh. u. Gynaek., 1926, xc, 89.

Pregnancy and diabetes; report of cases. B. D. BOWEN. Bull. Buffalo Gen. Hosp., Buffalo, N. York, 1926, iv, 51.

Is the glycosuria of pregnancy a diabetic disturbance of metabolism? D. ADLERSBERG and O. PORGES. Deutsche med. Wchnschr., 1926, lii, 653.

The treatment with insulin of acidosis associated with hyperemesis. H. SEIDL. Muenchen. med. Wchnschr., 1926, lxxiiii, 1471.

Clinical forms of toxæmia of pregnancy with complete or predominating symptoms of acidosis. A. TURENNE. Rev. argent. de obst. y ginec., 1926, x, 254.

The question of sterilization in anæmia of pregnancy simulating pernicious anæmia. A. REIST. Zentralbl. f. Gynaek., 1926, l, 1496.

Grave cardiopathy (gravidotoxic) in the first half of pregnancy. J. KREIS. Bull. Soc. d'obst. et de gynéc. de Par., 1926, xv, 524.

Chorea of pregnancy. LAURENTIE. Bull. Soc. d'obst. et de gynéc. de Par., 1926, xv, 527.

Icterus and pregnancy. T. HEYNEMANN. Zentralbl. f. Gynaek., 1926, l, 2181.

Renal function and pregnancy. R. KRAEUTER. Arch. f. Gynaek., 1926, cxxviii, 467.

Interruption of pregnancy in a case of pyelitis. A. FRUHINSHOLZ. Bull. Soc. d'Obst. et de gynéc. de Par., 1926, xv, 518.

Intractable vomiting and renal insufficiency. AUDEBERT and DALÉAS. Bull. Soc. d'obst. et de gynéc. de Par., 1926, xv, 529.

Pyelography and pyelonephritis of pregnancy. LEVY-SOLAL, MISRACHI, and SOLOMON. Gynéc. et obst., 1926, xiv, 218.

A case of hæmaturia with pregnancy in a retroverted uterus. JAMAL-UDDIN. Indian M. Gaz., 1926, lxi, 598.

Eclampsia. T. M. BURNS. Urol. & Cutan. Rev., 1926, xxx, 713.

A new theory of eclampsia. A. SZENES. Zentralbl. f. Gynaek., 1926, l, 2128.

The etiology and pathogenesis of eclampsia and the toxicosis of pregnancy. A. DIENST. Monatsschr. f. Geburtsh. u. Gynaek., 1926, lxxiv, 1.

Eclampsia and calcium. G. K. F. SCHULTZE. Zentralbl. f. Gynaek., 1926, l, 1463.

Studies in anæsthesia, anoxæmia, anhydræmia, and eclampsia, with certain deductions concerning the treatment of eclampsia. H. J. STANDER. Am. J. Obst. & Gynec., 1926, xii, 633.

Ascaridiasis and intractable vomiting of pregnancy. C. V. SUÁREZ. Bol. Soc. de obst. y ginec. de Buenos Aires, 1926, v, 403.

Twin pregnancy with degeneration of one ovum. S. E. BERMANN. Bol. Soc. de obst. y ginec. de Buenos Aires, 1926, v, 364.

Report of a case of acrania causing malpresentation and accidental hæmorrhage. A. J. FLEISCHER. Am. J. Obst. & Gynec., 1926, xii, 885.

Intra-uterine death of the fetus due to abnormalities of the umbilical cord. E. L. KING. Am. J. Obst. & Gynec., 1926, xii, 812.

The causation of still-birth and neonatal death. C. J. THOMSON. J. Obst. & Gynæc. Brit. Emp., 1926, xxxiii, 390. [301]

Placental transmission. IV. The protein fractions in fetal and maternal plasma. E. D. PLASS and C. W. MATTHEW. Am. J. Obst. & Gynec., 1926, xii, 847.

The protein-splitting ferments of the placenta. B. ARINSTEIN. Biochem. Ztschr., 1926, clxxi, 15.

Infarcts of the placenta. R. S. SIDDALL and F. W. HARTMAN. Am. J. Obst. & Gynec., 1926, xii, 683.

The secundines in relation to fetal pathology. H. D. FAIR. Med. J. & Rec., 1926, cxxiv, 670.

Premature separation of the normally inserted placenta at six months: cæsarean operation. SCHOCKAERT. Bull. Soc. d'obst. et de gynéc. de Par., 1926, xv, 545.

Recurring placenta prævia in a cordiform uterus. A. FORNERO. Clin. ostet., 1926, xxviii, 481.

The modern treatment of placenta prævia. J. C. LASCANO. Rev. argent. de obst. y ginec., 1926, x, 245.

Placenta marginada. M. OLASCOAGA. Rev. méd. d. Uruguay, 1926, xxix, 230.

A large placental polyp (placentoma). BELLANDI. Clin. ostet., 1926, xxviii, 487.

Gonorrhœa in pregnancy. E. V. DAVIS. Urol. & Cutan. Rev., 1926, xxx, 710.

The antiluetic treatment of pregnant women. J. FABRICIUS. Zentralbl. f. Gynaek., 1926, l, 2021.

Bismuth therapy and syphilis of the pregnant woman. M. L. PÉREZ. Bol. Soc. de obst. y ginec. de Buenos Aires, 1926, v, 387.

The recognition of lues during pregnancy and its clinical and social significance. W. BAER. Zentralbl. f. Gynaek., 1926, l, 2447.

The diagnosis and treatment of syphilis during pregnancy. NUERNBERGER. Beihefte z. Med. Klin., 1926, xxii, 121.

The prevention of abortion after operations on pregnant women. B. VON VÁRÓ. Zentralbl. f. Gynaek., 1926, l, 1964.

Experiences in 2,617 cases of abortion. A. STERNBERG. Deutsche med. Wchnschr., 1926, lii, 1548.

The indications for the induction of abortion. T. W. EDEN. Brit. M. J., 1926, ii, 237. [301]

Aspects to be considered for the termination of pregnancy before viability of the child. A. L. MCILROY. Brit. M. J., 1926, ii, 240. [301]

Slow and rapid methods of induction of abortion. J. D. BARRIS. Brit. M. J., 1926, ii, 241. [301]

Cardiac indications for termination of pregnancy before viability of the child. F. W. PRICE. Brit. M. J., 1926, ii, 242. [301]

The plea of insanity for the termination of pregnancy before the viability of the child. R. H. COLE. Brit. M. J., 1926, ii, 244. [301]

The ethical aspect of termination of pregnancy before viability of the child. H. H. EVERS. Brit. M. J., 1926, ii, 245. [301]

The basis for the therapeutic and prophylactic premature interruption of pregnancy. E. FREY. Schweiz. med. Wchnschr., 1926, lvi, 597.

The eugenic indication for abortion and sterilization in the presence of congenital malformations. F. A. VONNEGUT. Zentralbl. f. Gynaek., 1926, l, 2197.

The legalization of medically indicated abortion, with particular reference to the eugenic standpoint. KAHL. Arch. f. Frauenk. u. Konstitutionsforsch., 1926, xii, 50.

Sterilization and abortion from the legal standpoint. BAEHR. Monatsschr. f. Geburtsh. u. Gynaek., 1926, lxxiv, 88.

Methods of technique of anæsthesia in artificial abortion and other minor vaginal procedures. P. GELLERT. Zentralbl. f. Gynaek., 1926, l, 1127.

Therapeutic abortion in a tuberculous patient. ANDÉRODIAS and LESCARRET. Bull. Soc. d'obst. et de gynéc. de Par., 1926, xv, 479.

Abortion induced in a patient with pulmonary tuberculosis and non-ulcerating laryngeal tuberculosis. BOURSIER and FARGES. Bull. Soc. d'obst. et de gynéc. de Par., 1926, xv, 481.

Tolerance of the pregnant uterus in artificial abortion in double pregnancy in a double uterus. P. GARFUNKEL. Zentralbl. f. Gynaek., 1926, l, 2324.

On the interruption of pregnancy in the rat by the injection of ovarian follicular extract. M. G. SMITH. Bull. Johns Hopkins Hosp., Balt., 1926, xxxix, 203. **[304]**

Criminal abortion with soap solutions. O. POLANO. Muenchen. med. Wchnschr., 1926, lxxiii, 1317.

Death from septic endocarditis after criminal abortion more than four years before. VOELCKERS. Deutsche Ztschr. f. d. ges. gerichtl. Med., 1926, viii, 202.

Gas gangrene with embolism after abortion. E. STRASSMANN. Zentralbl. f. Gynaek., 1926, l, 1447.

Pregnancy complicated by fibroma; infection; abortion; hysterectomy. A. DELFOURD. Bull. Soc. d'obst. et de gynéc. de Par., 1926, xv, 516.

Axial torsion of the puerperal uterus caused by a fibroma. A. R. ENRIQUEZ and E. A. BOERO. Bol. Soc. de obst. y ginec. de Buenos Aires, 1926, v, 417.

Extirpation of ovarian tumors in pregnant women. B. VON VÁRÓ. Monatsschr. f. Geburtsh. u. Gynaek., 1926, lxxiv, 28.

Cancer of the rectum and pregnancy. H. KATZ and F. KASPAR. Arch. f. gynaek., 1926, cxxviii, 250.

Ileus and pregnancy. VON MIKULIZ-RADECKI. Muenchen. med. Wchnschr., 1926, lxxiii, 1352.

Ectopic pregnancy. C. CULBERTSON. Illinois M. J., 1926, l, 487.

Ectopic pregnancy. J. M. ALFORD. New Orleans M. & S. J., 1926, lxxix, 413.

The differential diagnosis of ectopic pregnancy. J. KOERNER. Muenchen med. Wchnschr., 1926, lxxiii, 985.

The umbilical phenomena in extra-uterine pregnancy. R. HOFSTAETTER. Arch. f. klin. Chir., 1926, cxl, 613.

Some cases of extra-uterine pregnancy. W. LIEBE. Zentralbl. f. Gynaek., 1926, l, 1150.

Peritonitis and extra-uterine pregnancy. G. H. SCHNEIDER. Monatsschr. f. Geburtsh. u. Gynaek., 1926, lxxiii, 304.

Tubal interstitial pregnancy of about two weeks; progressive peritoneal inundation. LAFFONT, HOUEL, and JAHIER. Bull. Soc. d'obst. et de gynéc. de Par., 1926, xv, 477.

A contribution on the treatment of tubal pregnancy. A. MANDELSTAMM. Zentralbl. f. Gynaek., 1926, l, 1659.

Referred pain in the shoulder in ruptured tubal pregnancy. W. C. DANFORTH. Am. J. Obst. & Gynec., 1926, xii, 883.

Early tubo-ovarian pregnancy. J. BATISWEILER. Monatsschr. f. Geburtsh. u. Gynaek., 1926, lxxiii, 77.

Ovarian pregnancy dating back forty-four years; lithopædion. E. SITTLER. Bull. Soc. d'obst. et de gynéc. de Par., 1926, xv, 522.

A case of abdominal pregnancy with intervention after term. LAURENTIE. Bull. Soc. d'obst. et de gynéc. de Par., 1926, xv, 534.

Secondary abdominal pregnancy with intra- and extra-uterine placenta—case report. D. H. DUFFIE. J. Michigan State M. Soc., 1926, xxv, 659.

Labor and Its Complications

A proof of Werboff's conclusions concerning the normal activity of the uterus during labor. N. TSCHISTIAKOFF. Monatsschr. f. Geburtsh. u. Gynaek., 1926, lxxiii, 258.

Remarks on Temesváry's work on the influence of extract of thymus on the action of the uterus and its practical application in obstetrics. H. KNAUS. Zentralbl. f. Gynaek., 1926, l, 1304.

The innervation of the uterus with reference to a case of labor in a patient with transverse paralysis of the cord. M. GERSTMANN. Monatsschr. f. Geburtsh. u. Gynaek., 1926, lxxiii, 253.

The constitution and first labor late in life. G. LUNDH. Zentralbl. f. Gynaek., 1926, l, 1582.

The new angular laminaria tent and its advantages. W. NIDEREHE. Med. Klin., 1926, xxii, 1192.

The Voorhees bag in the induction of labor, a criticism. A. L. MADDEN. Am. J. Obst. & Gynec., 1926, xii, 875.

Hypophyseal extract in obstetrics. VITAL AZA. Siglo méd., 1926, lxxiii, 332.

The use and misuse of pituitrin during labor. E. ESSEN-MOELLER. Svenska Laekartidningen, 1926, xxiii, 849.

The question of the prophylactic intravenous injection of hypophyseal extract during the third stage of labor. F. JESS. Zentralbl. f. Gynaek., 1926, l, 2440.

Artificial stimulation of pains with the glycerin bladder. F. A. SCHEFFZEK. Zentralbl. f. Gynaek., 1926, l, 2500.

Premature labor induced for the second time in a woman having previously undergone a cæsarean operation. L. VALLOIS. Bull. Soc. d'obst. et de gynéc. de Par., 1926, xv, 509.

Painless labor. K. STOLTE. Zentralbl. f. Gynaek., 1926, l, 2507.

Pain alleviation in obstetrics. O. PANKOW. Deutsche med. Wchnschr., 1926, lii, 1583, 1640.

Painless childbirth by the synergistic method. D. DEUTSCHMAN. Med. J. & Rec., 1926, cxxiv, 421.

The management of narcosis in child-birth. S. E. HUMPHREYS. Med. J. Australia, 1926, ii, 767.

The use of narcylen in obstetrics. H. FRANKEN. Zentralbl. f. Gynaek., 1926, l, 2455.

A case of reactive transitory stupor during labor. H. EUFINGER. Med. Klin., 1926, xxii, 1107.

Spontaneous delivery of the head with a transverse sagittal suture. R. BRUEHL. Zentralbl. f. Gynaek., 1926, l, 646.

The Pomeroy maneuver in the treatment of the persistent occiput-posterior position. H. ARANOW. N. York State J. M., 1926, xxvi, 961.

The Kjelland forceps. M. P. RUCKER. Virginia M. Monthr., 1926, liii, 580.

Slow dilatation and the use of Filho's caustic. H. VERMELIN. Bull. Soc. d'obst. et de gynéc. de Par., 1926, xv, 512.

Fœtal tumor obstructing delivery. L. W. DRYLAND. Brit. M. J., 1926, ii, 1223.

A case of dystocia due to fibroma prævia. LAFFONT. Bull. Soc. d'obst. et de gynéc. de Par., 1926, xv, 475.

Trial labor in the treatment of 477 cases of contracted pelves. H. BAILEY. Am. J. Obst. & Gynec., 1926, xii, 550. **[304]**

The mechanism of labor in contracted pelvis. P. SCHUMACHER. Arch. f. Gynaek., 1926, cxxix, 448.

Secondary pelvic contraction. R. BROOKE and F. COOK. Guy's Hosp. Rep., Lond., 1926, lxxvi, 467.

The treatment of contracted pelvis. M. TRAUGOTT. Jahresk. f. aerztl. Fortbild., 1926, xvii, 1.

The place of induction of premature labor in the treatment of contracted pelvis. J. B. BANISTER. Brit. M. J., 1926, ii, 519. **[305]**

Prophylactic median episiotomy during labor. D. VON OTT. Monatsschr. f. Geburtsh. u. Gynaek., 1926, lxxiv, 143.

Two cases of urgent symphyseotomy by a non-surgical method. D. E. V. ESQUIVEL. Semana méd., 1926, xxxiii, 985.

Two cases of temporary exteriorization of the uterus. LAFFONT, HOUEL, DUBOUCHER, JAHIER. Bull. Soc. d'obst. et de gynéc. de Par., 1926, xv, 473.

Sixteen cases of anterior median incisions of the cervix in the course of labor. AUDEBERT and GALY-GASPARROU. Bull. Soc. d'obst. et de gynéc. de Par., 1926, xv, 539.

Embryotomy on the living fetus. J. B. GONZÁLEZ. Semana méd., 1926, xxxiii, 1060.

Limitations for the cæsarean operation. A. B. SPALDING. Northwest Med., 1926, xxv, 526. **[305]**

Cæsarean section in the absence of uterine pains. B. LANGEN. Zentralbl. f. Gynaek., 1926, l, 2503.

A contribution to the technique of cæsarean section. F. J. MCCANN. J. Obst. & Gynæc. Brit. Emp., 1926, xxxiii, 426.

The technique of cæsarean section, with special reference to the lower uterine segment incision. J. M. MUNRO KERR. Am. J Obst. & Gynec., 1926, xii, 729.

Cæsarean section at The Johns Hopkins Hospital. J. W. WILLIAMS. Northwest Med., 1926, xxv, 519. **[305]**

The treatment of the third stage of labor in cervical cæsarean section. P. KLUMPER. Zentralbl. f. Gynaek., 1926, l, 2319.

A case of uterine rupture. J. PÉRY. Bull. Soc. d'obst. et de gynéc. de Par., 1926, xv, 482.

Rupture of the uterine scar at the beginning of the third labor in a woman twice cæsareanized. AUDEBERT and FOURNIER. Bull. Soc. d'obst. et de gynéc. de Par., 1926, xv, 533.

Is Credé's maneuver free from danger? R. DYROFF. Therap. d. Gegenw., 1926, lxvii, 255.

Uterine compression by Henkel's method in postpartum hæmorrhage. F. SPIRITO. Arch. di ostet. e ginec., 1926, 2 s. xiii, 261.

The possibility of causing harm by aortic compression. C. HASELHORST. Zentralbl. f. Gynaek., 1926, l, 1508.

Uterine inversion. A. ZUÑIGA. Bol. Soc. de cirug. de Chile, 1926, iv, 175.

Two cases of absolute shortness of the cord. V. RASCOL. Bull. Soc. d'obst. et de gynéc. de Par., 1926, xv, 544.

Appendicitis following labor; death. PÉRY and PRINCETEAU. Bull. Soc. d'obst. et de gynéc. de Par., 1926, xv, 493.

The sitting position following delivery. K. HEUSLER. Zentralbl. f. Gynaek., 1926, l, 1641.

Puerperium and Its Complications

The preventive aspects of postpartum care. C. J. MILLER. Am. J. Obst. & Gynec., 1926, xii, 856.

Breast feeding problems: I. Maternal. C. U. MOORE and H. G. DENNIS. J. Am. M. Ass., 1926, lxxxvii, 1970.

A new retractor for postpartum work. I. B. GREENE. Northwest Med., 1926, xxv, 658.

Postpartum hæmorrhages and their treatment. H. LORENZEN. Zentralbl. f. Gynaek., 1926, l, 1518.

Malta fever and the puerperium. A. IANNELLO. Rassegna internaz. di clin. e terap., 1926, vii, 480.

Aseptic postpartum cotyledon retention. H. VERMELIN. Bull. Soc. d'obst. et de gynéc. de Par., 1926, xv, 515.

The treatment of septic puerperal fever with large doses of alcohol by mouth. H. KUESTNER. Zentralbl. f. Gynaek., 1926, l, 1240.

The treatment of puerperal sepsis. S. J. GOODMAN. Ohio State M. J., 1926, xxii, 849. **[306]**

Newborn

The length and weight of the mature fetus in the light of statistical variations. E. WEHEFRITZ. Arch. f. Gynaek., 1926, cxxix, 227.

A comparative study of the development and physiology of identical twins. G. P. CROWDEN. Guy's Hosp. Rep., Lond., 1926, lxxvi, 379.

Observations on the blood of the newborn. E. HEATH. Bull. Buffalo Gen. Hosp., Buffalo, N. York, 1926, iv, 42.

The internal secretion at the beginning of life (before and after birth). E. THOMAS. 1926: Jena, Fischer.

The treatment of a child apparently born dead. F. KERMAUNER. Wien. klin. Wchnschr., 1926, xxxix, 1070.

Pressure softening of the skull in newborn children. H. ABELS. Wien. klin. Wchnschr., 1926, xxxix, 653.

Injury of the fetal skull by the uterine os. E. POECK. Zentralbl. f. Gynaek., 1926, l, 2316.

Recurring fetal hydrocephalus. D. IRAETA and E. HARGUINDEGUY. Semana méd., 1926, xxxiii, 934.

Intracranial hæmorrhage of the newborn. J. F. DICKS. Am. J. Obst. & Gynec., 1926, xii, 871.

Intracranial hæmorrhage of the newly born. C. J. BLOOM. New Orleans M. & S. J., 1926, lxxix, 435.

Intracranial hæmorrhage in the newly born. J. SIGNORELLI. New Orleans M. & S. J., 1926, lxxix, 448.

Meningeal hæmorrhage in the newborn. L. V. BLANCO and H. PAPERINI. J. Am. M. Ass., 1926, lxxxvii, 1261. **[306]**

The recognition of intracranial hæmorrhages in children born dead without autopsy. L. SCHOENHOLZ. Klin. Wchnschr., 1926, v, 1775.

Intestinal hæmorrhage in a newly born infant. B. SINGH. Indian M. Gaz., 1926, lxi, 598.

Some factors following transfusion in debilitated or premature infants. DALÉAS and GALY-GASPARROU. Bull. Soc. d'obst. et de gynéc. de Par., 1926, xv, 531.

Congenital ascites. FABREAU. Bull. Soc. d'obst. et de gynéc. de Par., 1926, xv, 487.

Anomaly of the sternum in a newborn child. L. VALLOIS and L. VIALLETON. Bull. Soc. d'obst. et de gynéc. de Par., 1926, xv, 501.

Report of a case of congenital malformation and arrested development of the colon. G. K. SIMS and H. L. MEYERS. Am. J. Obst. & Gynec., 1926, xii, 887.

New observations with reference to blue birth marks (Mongolian marks). M. ZARFL. Ztschr. f. Kinderh., 1926, xli, 356.

Miscellaneous

The need for the application of eugenic measures for the prevention of crime and for race betterment. C. F. DIGHT. J.-Lancet, 1926, n. s. xlvi, 557.

A plea for birth control of those who are physically or mentally unfit or financially unable to care for themselves and their children. R. E. JAMESON. J. Iowa State M. Soc., 1926, xvi, 535.

The problem of determination of sex: a collective review. L. CASLALDI. Rassegna internaz. di clin. e terap., 1926, vii, 554.

The problem of determination of sex. T. J. C. COMBES. Rev. de medicina, 1926, i, 92.

The work and study in the University Obstetrical Clinic. W. STOECKEL. Deutsche med. Wchnschr., 1926, lii, 1495.

The work of the French Maternity at Beyrouth. LAURENTIE. Bull. Soc. d'obst. et de gynéc. de Par., 1926, xv, 527.

The responsibility of the obstetrician. C. J. MILLER. South. M. J., 1926, xix, 882.

Why has the teaching of midwifery been so inefficient? C. BERKELEY. J. Obst. & Gynæc. Brit. Emp., 1926, xxxiii, 634.

A plea for drastic reforms in the teaching and practice of midwifery. G. W. THEOBALD. Lancet, 1926, ccxi, 633.

The nurse midwife in the Kentucky highlands. M. BRECKINRIDGE. Kentucky M. J., 1926, xxiv, 614.

A day in the life of a Leslie County midwife. M. BRECKINRIDGE. Kentucky M. J., 1926, xxiv, 615.

A medicolegal contribution with reference to obstetrical practice. M. HOFMEIER. Muenchen. med. Wchnschr., 1926, lxxiii, 743.

Clinical information based upon 1,000 consecutive obstetrical cases. W. E. LEVY. South. M. J., 1926, xix, 886.

The maneuver of Schatz-Unterberger in obstetrics. W. NETTESHEIM. Muenchen. med. Wchnschr., 1926, lxxiii, 1436.

Some common causes of maternal mortality. JELLETT. N. ZEALAND M. J., 1926, xxv, 312.

The reduction of the mortality and morbidity in childbirth. J. M. H. ROWLAND. J. Am. M. Ass., 1926, lxxxvii, 2158.

The value of glucose and insulin to the obstetrician and gynecologist. W. E. LEVY. Am. J. Obst. & Gynec., 1926, xii, 866.

Hæmorrhage in obstetrics and gynecology. E. GRAFF. Wien. med. Wchnschr., 1926, lxxvi, 1109.

Sepsis in obstetrics and gynecology. W. BENTHIN. Deutsche med. Wchnschr., 1926, lii, 1206.

Frequency and prophylaxis of syphilis at lying-in hospitals. A. P. RAMOS and M. L. PÉREZ. Semana méd., 1926, xxxiii, 945.

The nature of pseudo-iso-agglutination and its importance in the study of obstetrical serology. PELLISSIER and FALGAIROLLES. Bull. Soc. d'obst. et de gynéc. de Par., 1926, xv, 498.

Concerning immune substances in the colostrum. E. HAINISS. Deutsche med. Wchnschr., 1926, lii, 1552.

GENITO-URINARY SURGERY

Adrenal, Kidney, and Ureter

The function of the suprarenal capsule. C. VIALE, T. COMBES, A. BRUNO, DI LEO LIRA, and E. TURCATTI. Rev. Soc. argent. de biol., 1926, ii, 528.

Notes on cases of adrenal insufficiency. MURRAY. N. Zealand M. J., 1926, xxv, 307.

Suprarenal hæmorrhage: report of a case. E. B. CLARK. California & West. Med., 1926, xxv, 751.

Neuroblastoma of the adrenal gland (Hutchinson type): report of a case. M. LEDERER. J. Cancer Research, 1926, x, 377.

Severe renal trauma with rupture of the renal artery: absence of hæmaturia. A. H. GREENWOOD. Canadian M. Ass. J., 1926, xvi, 1500.

Further studies on the transplantation of the kidney. C. S. WILLIAMSON. J. Urol., 1926, xvi, 231. **[307]**

Observations on reduplication of kidney pelves and ureters, with a case report. F. L. SCHUMACHER. Radiology, 1926, vii, 475.

Hypernephroma in a horseshoe kidney. H. F. WECHSLER. Endocrinology, 1926, x, 493.

Horseshoe kidney and operations for horseshoe kidney. A. FISCHER. Ztschr. f. urol. Chir., 1926, xx, 257.

New investigations of the pathological significance of accessory renal vessels. ANDLER. Zentralbl. f. Chir., 1926, liii, 1009.

The technique of the acid-alkali functional test of pathological kidneys. G. VON PANNEWITZ. Ztschr. f. urol. Chir., 1926, xx, 19.

Testing renal function with urea in surgery. RADÓ, BÉLA, and DEUTSCH. Ztschr. f. urol. Chir., 1926, xx, 191.

Functional insufficiency of the kidney after ureterocystoneostomy. CATHELIN. Bull. et mém. Soc. d. chirurgiens de Par., 1926, xviii, 594.

The functional results of retention in the renal pelvis. B. FEY. Arch. urol. de la clin. de Necker, 1926, v, 93. **[307]**

Contributions to our knowledge of diseases of the kidney and liver. L. G. ROWNTREE. Canadian M. Ass. J., 1926, xvi, 1306.

Drainage as a factor in renal disease. G. L. HUNNER. Surg., Gynec. & Obst., 1926, xliii, 615.

Nephrosis. C. A. LILLY. J. Kansas M. Soc., 1926, xxvi, 381.

Hydronephrosis. V. C. HUNT. Surg. Clin. N. Am., 1926, vi, 1153.

Nephrectomy for pyonephrosis in nurslings, with a contribution upon the pathogenesis of pyelonephritis. F. BRUENING. Klin. Wchnschr., 1926, v, 1652.

Closed renal tuberculosis: with a report on five cases. H. P. W. WHITE. Lancet, 1926, ccxi, 1310.

Gummatous sclerosing syphilis of the kidney. F. R. RUIZ. Rev. de medicina, 1926, i, 77.

Pyelitis. H. BOEMINGHAUS. Ergebn. d. Chir. u. Orthop., 1926, xix, 583.

Etiology, symptoms, and diagnosis of pyelitis. J. M. ANDERS. Atlantic M. J., 1926, xxx, 170.

Renal hæmaturia. C. MAZER. Atlantic M. J., 1926, xxx, 167.

Partial nephrectomy for hydatid cyst of the kidney lying in the pelvis. A. BENGOLEA. Rev. de cirug., Buenos Aires, 1926, v, 466.

Tumors of the renal capsule. K. HASLINGER. Ztschr. f. urol. Chir., 1926, xx, 89.

Papillary tumors of the renal pelvis. K. HASLINGER. Ztschr. f. urol. Chir., 1926, xx, 77.

The clinical aspects and diagnosis of renal calculi which are permeable to the X-rays. Z. KAIRIS. Ztschr. f. urol. Chir., 1926, xx, 66.

Uric acid and uratic stones in the kidney—uric acid showers and their diagnosis. E. BEER. Surg., Gynec. & Obst., 1926, xliii, 436. **[310]**

Nephrotomy: its importance and the method of preventing its dangers; a clinical and animal experimental study. A. ROSENO. Ztschr. f. urol. Chir., 1926, xx, 96, 127.

Postoperative hæmorrhage following nephrotomy and its prevention. A. ROSENO. 50 Tag. d. deutsch. Ges. f. Chir., Berlin, 1926. **[311]**

Resection of the kidney for stone. A. J. SCHOLL. California & West. Med., 1926, xxv, 341.

Renal arterial variations and extraperitoneal abdominal nephrectomy. B. LIPSHUTZ and C. HOFFMAN. Ann. Surg., 1926, lxxxiv, 525. **[311]**

Renal affections operated upon through Louis Bazy's incision. A. ZAVADZKI. Bull. et mém. Soc. nat. de chir., 1926, lii, 866.

Reduplication of the ureter with various anomalies of the ureteral opening in the bladder. C. H. LUDOWIGS. Ztschr. f. urol. Chir., 1926, xx, 203.

The report of a case of bifid ureter with partial urone phrosis. C. COLUCCI. Policlin., Rome, 1926, xxxiii, sez. chir., 447.

The pathogenesis and treatment of ureterocele. J. GOTTLIEB. Ztschr. f. urol. Chir., 1926, xx, 1.

Ureteral obstruction. A. C. GILBERT. Texas State J. M., 1926, xxii, 522.

The removal of deeply situated ureteral calculi. E. JOSEPH. 50 Tag. d. deutsch. Ges. f. Chir., Berlin, 1926. [311]

Large ureteral calculi—case report. W. T. BRIGGS. Urol. & Cutan. Rev., 1926, xxx, 708.

Empyema of the stump of the ureter. R. FRONSTEIN. Ztschr. f. urol. Chir., 1926, xx, 183.

The end-results of a ureteral anastomosis. MCEACHERN. Ann. Surg., 1926, lxxxiv, 894.

Experimental studies on crushing of the ureter with the hæmostatic forceps. L. TORRACA. Ann. ital. di chir., 1926, v, 651.

Bladder, Urethra, and Penis

A combination irrigating and illuminating stand for cystoscopic work. G. F. MCKIM and P. G. SMITH. Urol. & Cutan. Rev., 1926, xxx, 720.

A ureterotome for use with the cystoscope. J. N. V. VEER and J. E. HESLIN. Urol. & Cutan. Rev., 1926, xxx, 720.

Bladder symptoms from congenital deformities with associated nerve lesions: report of three cases. F. H. COLBY. Boston M. & S. J., 1926, cxcv, 804. [312]

A giant calculus of the bladder. G. E. SERRANO. Rev. med.-quirurg. hospitales, 1926, ii, 350.

The first case of tricomoniasis of the bladder described in Peru. E. ESCOMEL. Crón. méd., Lima, 1926, xliii, 185.

Neuromuscular dysfunction of the bladder as a cause of chronic pyelitis in childhood. H. F. HELMHOLZ. Am. J. Dis. Child., 1926, xxxii, 682.

The uretero-vesical opening demonstration of a new fulgurating incisor. N. S. MOORE. J. Urol., 1926, xvi, 541.

A new method of electrically excising obstructing bladder neck contractures and bars. C. W. COLLINGS. J. Urol., 1926, xvi, 545.

Resection of obstructions at the vesical orifice; new instruments and a new method. M. STERN. J. Am. M. Ass., 1926, lxxxvii, 1726.

Intramural carcinoma of the dome of the bladder. J. D. BARNEY. J. Urol., 1926, xvi, 369.

The treatment and prognosis of malignant tumors of the bladder. SIMON. Zentralbl. f. Chir., 1926, liii, 1011.

Ureteral transplantation in bladder carcinoma. A. L. CHUTE. J. Am. M. Ass., 1926, lxxxvii, 1613.

Deep roentgen-ray therapy in the treatment of carcinoma of the bladder. C. A. WATERS. J. Am. M. Ass., 1926, lxxxvii, 1618.

The clinical picture and treatment of reduplication of the urethra with epispadias. A. RITTER. Ztschr. f. urol. Chir., 1926, xx, 5.

A case of diphtheritic urethritis in a man. F. F. IMIANITOFF. Bruxelles-méd., 1926, vi, 1575.

Congenital stenosis of the urethra. MIRALLÈS VILA. Bull. et mém. Soc. d. chirurgiens de Par., 1926, xviii, 589.

Epithelioma of the penis. W. W. GIBBS. J. Nat. M. Ass., 1926, xviii, 200.

A massive papilloma of the penis resembling a carcinoma. F. J. PARMENTER. Bull. Buffalo Gen. Hosp., Buffalo, N. York, 1926, iv, 49.

Clinical and statistical study of carcinoma of the penis. V. FOEDERL. Deutsche Ztschr. f. Chir., 1926, cxcviii, 207.

Genital Organs

Twenty cases of prostatic hypertrophy treated with the roentgen rays. A. HELSTED. Zentralbl. f. Chir., 1926, liii, 1149.

The use of Payr's pepsin-Pregl solution in urinary retention due to pathological changes in the prostate. S. HOFFHEINZ. Deutsche Ztschr. f. Chir., 1926, cxcviii, 231.

Recurrences following prostatectomy by the method of Freyer. R. FRONSTEIN and G. MESCHEBOWSKI. Ztschr. f. urol. Chir., 1926, xx, 222. [312]

Metastasis in carcinoma of the prostate, with the report of a case showing early and late changes. J. SPROULL. Am. J. Roentgenol., 1926, xvi, 565.

Abnormal descent of the testicle. C. G. BURDICK and B. L. COLEY. Ann. Surg., 1926, lxxxiv, 867.

The interstitial cells and the cells of Sertoli of the testis. V. DIAMARE. Rassegna internaz. di clin. e terap., 1926, vii, 453.

Testicular transplants in man. R. SPURR. Crón. méd., Lima, 1926, xliii, 209.

The importance of the site of testicular transplants: an experimental study. C. TRINCHERA. Policlin., Rome, 1926, xxxiii, sez. chir., 484.

Hermaphrodism. MATHIEU, LAPOINTE, TUFFIER, and OMBRÉDANNE. Bull. et mém. Soc. nat. de chir., 1926, lii, 865.

The treatment of phagedenic ulcerations of the genitals with intravenous administration of antimony and potassium tartrate. J. KINGSBURY and S. M. PECK. J. Am. M. Ass., 1926, lxxxvii, 1900.

Miscellaneous

Urology—a specialized entity. G. R. O'CROWLEY. J. Urol., 1926, xvi, 411.

Hydrogen-ion studies on various secretions of the urogenital apparatus. A. RANDALL and M. MUSCHAT. J. Urol., 1926, xvi, 515.

Frequency of micturition. T. E. HAMMOND. Brit. M. J., 1926, ii, 1056.

A device for collecting urine from suprapubic incisions. E. G. BALLENGER. J. Urol., 1926, xvi, 539.

Pathological relations between the urinary and digestive apparatus. I. S. COVISA. Rev. méd. de Sevilla, 1926, xliv, 1.

Pyelovenous back flow. F. HINMAN and F. H. REDEWILL. J. Am. M. Ass., 1926, lxxxvii, 1287. [313]

Retention of urine in children with and without demonstrable cause. F. J. PARMENTER and C. LEUTENEGGER. Am. J. Dis. Child., 1926, xxxii, 692.

Urinary obstructions in childhood. C. G. MIXTER. Ann. Surg., 1926, lxxxiv, 533. [313]

The pathogenesis of oxaluria. E. LOMBARDI. Riforma med., 1926, xlii, 867.

Hæmaturia. P. J. KAHLE. New Orleans M. & S. J., 1926, lxxix, 422.

A case of hæmaturia. E. L. YOUNG, JR. Boston M. & S. J., 1926, cxcv, 1246.

Hæmaturia of unusual cause. J. D. BARNEY. Boston M. & S. J., 1926, cxcv, 1068.

Adynamic ileus in urology. R. M. LECOMTE. Virginia M. Month., 1926, liii, 596.

Parasitic infection of the urinary tract. E. C. MEKIE. Edinburgh M. J., 1926, n.s. xxxiii, 708.

Program of the antivenereal campaign in Peru. G. ALARCO. Crón. méd., Lima, 1926, xliii, 245.

Social diseases in children. V. GROSSI. Arch. Soc. méd. de Valparaíso, 1926, i, 51.

Gonorrhœa. L. W. HARRISON. Brit. M. J., 1926, ii, 1057.

The diagnosis and treatment of gonorrhœa. E. LAMBKIN. Med. Press, 1926, n.s. cxxii, 491.

Intravenous chemotherapy in gonorrhœa. F. E. GRIMALDI and R. DE SURRA CANARD. Semana méd., 1926, xxxiii, 1017.

Urinary antiseptics. G. G. REINLE and E. S. DEPUY. California & West. Med., 1926, xxv, 744.

Errors of position of the roentgenographic picture of urinary calculosis. ISELIN. Bull. et mém. Soc. d. chirurgiens de Par., 1926, xviii, 582.

The roentgen ray in urology. J. C. SARGENT. Radiology, 1926, vii, 480.

The clinical aspects of surgical diseases of the urinary tract in children. A. HYMAN. J. Urol., 1926, xvi, 259. [314]

Malignant growths of the lower urinary tract. D. W. MACKENZIE. Boston M. & S. J., 1926, cxcv, 811. [315]

The results of X-ray therapy of malignant growths of the urinary tract. L. T. MANN. Surg., Gynec. & Obst., 1926, xliii, 529. [315]

A review of urological surgery. A. J. SCHOLL, E. S. JUDD, L. D. KEYSER, G. S. FOULDS, and others. Arch. Surg., 1926, xiii, 913.

A discussion on spinal anæsthesia in urology. H. W. MARTIN and R. E. ARBUTHNOT. J. Am. M. Ass., 1926, xxxvii, 1723.

The value of transillumination in surgery of the urinary tract. V. J. O'CONOR. J. Urol., 1926, xvi, 459.

SURGERY OF THE BONES, JOINTS, MUSCLES, TENDONS

Conditions of the Bones, Joints, Muscles, Tendons, Etc.

Injuries due to sport. WIEWIOROWSKI. Zentralbl. f. Chir., 1926, liii, 1265.

The relations of the so-called function of bone to bone regeneration. H. BURCKHARDT. Deutsche Ztschr. f. Chir., 1926, cxcviii, 21.

Bernhard Haines' studies of bone regeneration: his life and his time: K. VOGELER, H. WALTER, E. REDENZ, and B. MARTIN. 1926: Berlin, Springer.

The metamorphosis of transplanted bones as they appeared in the roentgen picture. GULEKE. Arch. f. klin. Chir., 1926, cxli, 325.

Histological studies of experimentally produced pseudarthroses. A. SCHMIDT. Beitr. z. klin. Chir., 1926, cxxxvi, 463.

Osteitis fibrosa. G. SCHMORL. Zentralbl. f. allg. Path. u. path. Anat., 1926, xxxvii, 71, 134.

Osteitis fibrosa. R. WANKE. Beitr. z. klin. Chir., 1926, cxxxvi, 664.

Cystic fibrous osteitis of malignant evolution. NOVÉ-JOSSERAND. Lyon chir., 1926, xxiii, 604.

The pathogenesis of the osteitis fibrosa of Recklinghausen. E. LOESER. Zentralbl. f. allg. Path. u. path. Anat., 1926, xxxvii, 91, 134.

Typhoid osteitis and periosteitis. D. Y. KEITH and J. P. KEITH. J. Am. M. Ass., 1926, lxxxvii, 2145.

The treatment of acute hæmatogenous infectious osteomyelitis. T. P. KRASNOBAJEV. Novyj chirurgičeskij archiv, 1925, viii, 354. [316]

Osteomyelitis followed by calcareous resorption and deposit in the soft tissues. F. D. WEIDMAN. Atlantic M. J., 1926, xxx, 163.

Tuberculous osteomyelitis. PEABODY. Ann. Surg., 1926, lxxxiv, 894.

The present status of the study of tuberculosis of the bones and joints. BUECHERT. Aerztl. Monatsschr., 1926, p. 17, 51.

Osteopetrosis. R. G. KARSHNER. Am. J. Roentgenol., 1926, xvi, 405.

Some reflections on the pathogenesis of rickets. LAURENTIE. Bull. Soc. d'obst. et de gynéc. de Par., 1926, xv, 536.

Effects of diet on rickets. J. V. GREENEBAUM, T. K. SELKIRK, F. A. OTIS, and A. G. MITCHELL. J. Am. M. Ass., 1926, lxxxvii, 1973.

Essential bone cysts. A. JOHOW. Bol. Soc. de cirug. de Chile, 1926, iv, 178.

Giant cell tumors of the long bones. E. GOLD. Arch. f. klin. Chir., 1926, cxl, 763.

Massage and remedial exercises in bone and joint diseases. W. H. TRETHOWAN. Guy's Hosp. Rep., Lond., 1926, lxxvi, 433.

A further contribution on the question of congenital hydrocephalus associated with chondrodystrophy. G. B. GRUBER. Zentralbl. f. allg. Path. u. path. Anat., 1926, xxxvii, 315.

The mechanism of acute bacterial infection of a joint. A. O. WILENSKY. Arch. Surg., 1926, xiii, 895.

The influence of focal infection and the pathology of arthritis: results of experiments. R. PEMBERTON, F. A. CAJORI, and C. Y. CROUTER. J. Am. M. Ass., 1926, lxxxvii, 2148.

A consideration of joint affections with special reference to acute rheumatic fever. L. G. HEYN. Cincinnati J. M., 1926, vii, 530.

Gonococcic arthritis. D. DE FRENELLE. Paris chir., 1926, xviii, 225.

Chronic infectious arthritis with multiple ankylosis of joints and complete disability. H. W. MEYERDING. Surg. Clin. N. Am., 1926, vi, 1301. [316]

Arthritis deformans. H. C. ADAMS. Med. J. Australia 1926, ii, 753.

Clinical significance of hyperglycæmia in arthritis deformans. J. D. BOYD and M. V. NELSON. J. Iowa State M. Soc., 1926, xvi, 528.

A contribution on the pathological physiology of the joints with reference to arthritis deformans. P. SEELIGER. Deutsche Ztschr. f. Chir., 1926, cxcviii, 11.

A contribution on the diagnosis and treatment of erysipelatous arthritis. F. BARTH. Beitr. z. klin. Chir., 1926, cxxxvii, 170.

Articular staphylomycosis. G. BOLOGNESI. Chir. d. organi di movimento, 1926, xi, 65.

The pathological physiology of the joints; the formation of joint mice. SEELIGER. 50 Tag. d. deutsch. Ges. f. Chir., Berlin, 1926. [316]

The fate of effusions of blood in the joints. P. SEELIGER. Klin. Wchnschr., 1926, v, 1616.

Metastatic intramuscular gonococcal abscess. B. NEWBURGER. Ann. Surg., 1926, lxxxiv, 879.

Humerus varus. F. SATTA. Chir. d. organi di movimento, 1926, xi, 57.

Ischæmic contracture. W. LOEHR. 50 Tag. d. deutsch. Ges. f. Chir., Berlin, 1926. [317]

Fracture of the ulnar diaphysis followed by radio-ulnar synostosis. PATEL and ALBERTIN. Lyon chir., 1926, xxiii, 592.

Syphilitic osteomyelitis of the ulna: acute arthritis of the elbow. C. VILLARÁN. Crón. méd., Lima, 1926, xliii, 145.

The digitated stump in amputation of the forearm. T. GIOIA. Semana méd., 1926, xxxiii, 1073.

A new method of curing ganglia of the wrist joint. J. J. GINZBURG. Zentralbl. f. Chir., 1926, liii, 1176.

Infected hand followed by loss of power in the extensors of the fingers and thumbs. G. G. CORBETT. Canadian M. Ass. J., 1926, xvi, 1502.

Arthritis deformans of the articulations of the transverse processes of the ribs. A. BERNSTEIN. Arch. f. klin. Chir., 1926, cxli, 419.

Alleged back injuries. L. G. HARNEY. Illinois M. J., 1926, l, 483.

A consideration of backache from the orthopedic standpoint. W. A. COCHRANE. Med. Press, 1926, n.s. cxxii, 529.

Lumbago. A. MUELLER. 1926: Bonn, Marcus and Weber.

Pain due to lumbosacral bone changes. G. PICCINNINO. Rassegna internaz. di clin. e terap., 1926, vii, 464.

The development of the scoliotic vertebral column. PORT. Ztschr. f. orthop. Chir., 1926, xlvii, 179, 203.

X-ray clinic on lesions of the vertebræ. P. M. HICKEY. Ann. Clin. Med., 1926, v, 95. **[317]**

Infectious spondylitis and growth spondylitis: a contribution on "false Pott's disease." J. MONTAGNE. Presse méd., Par., 1926, xxxiv, 1242.

Spondylitis of unknown etiology simulating typhoid spine. S. C. WOLDENBERG. Illinois M. J., 1926, l, 463.

Anatomical and postural variations of the lumbosacral spine. E. H. SKINNER and E. R. DEWEESE. J. Missouri State M. Ass., 1926, xxiii, 439.

Echinococcus disease of the pelvic bones. A. ANGELI. Chir. d. organi di movimento, 1926, xi, 25.

Diseases of the hip joint. A. MITCHELL. Brit. M. J., 1926, ii, 1056.

The laws of inflammatory contracture of the joints, illustrated by the hip joint. F. FRIEDLAENDER. Deutsche Ztschr. f. Chir., 1926, cxcvi, 121.

Non-tuberculous hips in children simulating tuberculosis. C. B. FRANCISCO. J. Missouri State M. Ass., 1926, xxiii, 385.

Osteo-articular amœbiosis of the hip. F. L. NIÑO and A. MASUCCI. Bol. inst. de clín. quir., 1926, ii, 280.

Late diseases of the hip joint following reduced congenital luxations. A. SCHANZ. Ztschr. f. aerztl. Fortbild., 1926, xxiii, 593.

Non-tuberculous coxitis in the young. H. A. T. FAIRBANK. Brit. M. J., 1926, ii, 828.

The pathogenesis of acetabular protrusion due to coxitis. R. BENDA. Arch. f. Gynaek., 1926, cxxix, 186.

A case of chronic arthritis of the hip. N. ALLISON. Boston M. & S. J., 1926, cxcv, 1068.

Osteo-arthritis of the hip joint treated by vaccines. H. W. CROWE. Brit. M. J., 1926, ii, 834.

Curative attempts in spontaneous diseases of the hip with Bayer-205 or naganol. F. ROSENBUSCH and J. ACOSTA. Bol. inst. de clín. quir., 1926, ii, 249.

Quantitative determination of Trendelenburg's phenomena of the hip joint. H. KNORR. Ztschr. f. orthop. Chir., 1926, xlvii, 365.

Surgical lesions of the hip joint. M. S. HENDERSON. Surg. Clin. N. Am., 1926, vi, 1283.

Coxa vara following bone cyst of the greater trochanter with pathological fracture. H. W. MEYERDING. Surg. Clin. N. Am., 1926, vi, 1301.

The traumatic development of coxa vara of adolescence from the standpoint of the estimation of the effect of injury. SAALMANN. Monatsschr. f. Unfallheilk. u. Versicherungsmed., 1926, xxxiii, 108.

The development of coxa valga. HACKENBROCH. Zentralbl. f. Chir., 1926, liii, 2411.

A new theory about the pathogenesis of coxa plana and other manifestations of "local dyschondroplasia." P. G. K. BENTZON. Brit. J. Radiol., 1926, xxxi, 439.

A contribution to our knowledge of Perthes' and Koehler's disease. KONJETZNY. 50 Tag. d. deutsch. Ges. f. Chir., Berlin, 1926. **[319]**

Inflections and acquired deformities of the upper end of the femur. GABRIELLE. Lyon chir., 1926, xxiii, 601.

The blood vessels in the upper end of the femur and their relation to pathological processes. A. NUSSBAUM. Beitr. z. klin. Chir., 1926, cxxxvii, 332.

Femoral osteochondritis of adolescents and its sequelæ; epiphyseal separation of the hip. I. BALENSWEIG. Surg., Gynec. & Obst., 1926, xliii, 604.

An anatomical and roentgenological study of the ossification centers of the knee in a newborn child. R. JARDIN. Gynéc. et obst., 1926, xiv, 240.

A singular type of genu varum. HACKENBROCH. Ztschr. f. orthop. Chir., 1926, xlvii, 391, 405.

The prevention of fixation ankylosis particularly of the knee joint. H. RICHTER. Zentralbl. f. Chir., 1926, liii, 1938.

A case of spontaneous fracture of the patella complicating primary periosteal sarcoma. C. F. BIANCHETTI. Chir. d. organi di movimento, 1926, xi, 46.

The pathology of the menisci of the knee. D. DE FRENELLE. Paris chir., 1926, xviii, 217.

The treatment of sprained ankles. A. B. LIGHT. Therap. Gaz., 1926, 3 s. xlii, 844.

The importance of the shoe in the development and treatment of deformities of the foot. H. MEYER. Ergebn. d. Chir. u. Orthop., 1926, xix, 475.

A contribution on Koehler's disease. A. MOSENTHAL. Med. Klin., 1926, xxii, 1256.

Deutschlaender's inflammatory disease of the metatarsus. R. BITTROLFF. Zentralbl. f. Chir., 1926, liii, 1634.

Remarks on congenital metatarsus varus and its treatment. E. BERGMANN. Arch. f. klin. Chir., 1926, cxli, 472.

The treatment of congenital talipes. A. W. ADAMS. Brit. M. J., 1926, ii, 1053.

The painful heel. T. TOEPEL. J. Med. Ass. Georgia, 1926, xv, 460.

Calcaneal epiphysitis. U. RACCA. Gior. batteriol. e immunol., 1926, i, 491.

A case of marked ossifying periostitis of the calcaneus after operation for a calcaneal spur, a subcalcaneal bursa, and an Achilles bursa. C. MAU. Zentralbl. f. Chir., 1926, liii, 2462.

Painless calcaneal spurs. C. I. ALLENDE. Rev. de cirug., Buenos Aires, 1926, v, 470.

The results of the roentgen treatment of bone and joint tuberculosis in the region of the foot. O. JUENGLING. Acta radiol., 1926, vii, 14. **[319]**

Surgery of the Bones, Joints, Muscles, Tendons, Etc.

The importance of the spongiosa in bone transplantation. A. BÁRON. Zentralbl. f. Chir., 1926, liii, 2332.

Investigations of the suitability of the intermediate mountainous areas for the treatment of surgical diseases of the bones and joints. H. TICHY. Beitr. z. klin. Chir., 1926, cxxxvii, 217.

The treatment of localized and generalized osteitis fibrosa. F. MANDL. Wien. klin. Wchnschr., 1926, xxxix, 1046.

The treatment of acute osteomyelitis. S. JOHANSSON. Zentralbl. f. Chir., 1926, liii, 1126.

The treatment of acute and chronic osteomyelitis. H. SCHILLING. Zentralbl. f. Chir., 1926, liii, 1127.

The use of the Italian method of treatment in four cases of open bone and joint tuberculosis. P. LAĚNÝ. Bratislavské lekárske listy, 1926, v, 742.

The combined treatment of bone and joint tuberculosis. O. VULPIUS. Fortschr. d. Med., 1926, xliv, 589.

A new method of osteosynthesis. T. MAYEDA. Deutsche Ztschr. f. Chir., 1926, cxcvi, 311.

The treatment of pseudarthroses. M. ZONDEK. Zentralbl. f. Chir., 1926, liii, 1234.

The technique of bone suture. G. MAGNUS. Zentralbl. f. Chir., 1926, liii, 2514.

The surgical treatment of gonorrhœal arthritis. E. ONDIVIELA. Ars med., 1926, ii, 229.

The treatment of arthritis deformans. J. W. HOETS. Med. J. Australia, 1926, ii, 755.

New viewpoints in arthroplasty. S. KOFMANN. Zentralbl. f. Chir., 1926, liii, 1125.

Plastic operations on the large ball and socket joints. PAYR. 50 Tag. d. deutsch. Ges. f. Chir., Berlin, 1926. **[320]**

The use of fascia for the re-enforcement of relaxed joints. G. E. BENNETT. Arch. Surg., 1926, xiii, 655.

Experimental use of artificial tendons. H. MEYER. Ztschr. f. orthop. Chir., 1926, xlvii, 237, 269.

The regeneration of the tendons and the tendon sheaths and the treatment of divided tendons. E. J. NAERVI. Zentralbl. f. Chir., 1926, liii, 1151.

Experimental investigations on the prevention of adhesions in the transplantation of tendons and the production of a rigid connective tissue by chemical means in the treatment of orthopedic conditions. P. PITZEN. Ztschr. f. orthop. Chir., 1926, xlvii, 385.

Partial excision of the scapula for enchondroma. JACKSON. Ann. Surg., 1926, lxxxiv, 894.

Resection of the elbow for traumatic ankylosis. E. UGARTE and C. CORTES. Bol. Soc. de cirug. de Chile, 1926, iv, 171.

The late results of resection of the elbow. J. SÉNÈQUE. Presse méd., Par., 1926, xxxiv, 1351.

Tendon transplantation for radial paralysis. NOVÉ-JOSSERAND. Lyon chir., 1926, xxiii, 603.

Late results of operation for wry neck. J. NUSSBAUM. Beitr. z. klin. Chir., 1926, cxxxvi, 573.

The operative splinting of the diseased vertebral column by bone transplantation. A. HENLE and E. HUBER. Ergebn. d. Chir. u. Orthop., 1926, xix, 349.

Resection of the first rib. F. BERNDT. Deutsche Ztschr. f. Chir., 1926, cxcvii, 71.

Reconstruction operation on the hip. J. S. SPEED. J. Am. M. Ass., 1926, lxxxvii, 1631.

Remarks prefatory to a cinematographic presentation of late results of the reconstruction operation. A. WHITMAN. J. Bone & Joint Surg., 1926, viii, 803. **[321]**

The Whitman reconstruction operation. COLONNA. Ann. Surg., 1926, lxxxiv, 894.

The correction of deformity in quiescent disease of the hip. L. C. ABBOTT. J. Am. M. Ass., 1926, lxxxvii, 1095. **[321]**

A new method of resection of the hip. R. D. SARASOLA and V. S. PERPIÑA. Zentralbl. f. Chir., 1926, liii, 2329.

The extirpation of the femur with plastic substitution of the leg by Sauerbruch's method. FRUEND. Deutsche Ztschr. f. Chir., 1926, cxcvi, 241.

Clinical experiences with Sauerbruch's plastic substitution of the leg for the femur. F. KRAMPF. Deutsche Ztschr. f. Chir., 1926, cxcvi, 246.

Two cases of coxa vara, the one treated by re-infraction, the other by subtrochanteric osteotomy. DELAHAYE and SORREL. Bull. et mém. Soc. nat. de chir., 1926, lii, 855.

Transpatellar exposure of the knee joint. BÉRARD. Lyon chir., 1926, xxiii, 654.

Excision of the internal semilunar cartilage. O'CONOR. Ann. Surg., 1926, lxxxiv, 894.

Rupture of the antero-external crucial ligament of the knee: Hey-Groves' operation: recovery. BAUMGARTNER. Bull. et mém. Soc. nat. de chir., 1926, lii, 879.

The late result of the treatment of a vicious ankylosis of the knee. TIXIER. Lyon chir., 1926, xxiii, 612.

A case of arthrodesis of the knee. V. SIMEONI. Ann. ital. di chir., 1926, v, 698.

Lateral displacement of the fibula in the presence of large tibial defects. O. STRACKER. Zentralbl. f. Chir., 1926, liii, 2132.

Osteotomy and suture of the tibia in severe rachitic curvature of the leg. R. MINERVINI. Chir. d. organi di movimento, 1926, xi, 84.

Two cases of astragalectomy for vicious consolidation of bimalleolar fractures. AUTEFAGE and GERNEZ. Bull. et mém. Soc. nat. de chir., 1926, lii, 903.

The treatment of flat-foot by exeresis. BUCHOLZ. Ztschr. f. orthop. Chir., 1926, xlvii, 332, 342.

Flat-foot and its treatment, with particular reference to the operative method of Ernst Mueller. K. VON DITTRICH. Deutsche Ztschr. f. Chir., 1926, cxcvi, 194.

Late results of the operative treatment of hallux valgus. H. PICK. Arch. f. orthop. u. Unfall-Chir., 1926, xxiv, 251.

Fractures and Dislocations

A plea for better care of fractures. A. H. SMITH. Internat. J. Med. & Surg., 1926, xxxix, 488.

The fundamental principles in the treatment of fractures. R. CAROTHERS. Ohio State M. J., 1926, xxii, 1025.

The operative treatment of fractures. C. L. SCUDDER. Boston M. & S. J., 1926, cxcv, 1187. Rhode Island M. J., 1926, ix, 153.

The importance of nerve block anæsthesia in the treatment of fractures and dislocations. W. MUELLER. Med. Klin., 1926, xxii, 327.

A short résumé of fracture experiences at the Norton Company. W. I. CLARK. Boston M. & S. J., 1926, cxcv, 1064.

The fate of the fragment; a study of comminuted fractures. F. E. CLOUGH. Surg., Gynec. & Obst., 1926, xliii, 790.

The treatment of spiral fractures. A. B. MITCHELL. Brit. M. J., 1926, ii, 1048.

The method of action of adhesive plaster tension in the management of fractures. V. DUMPERT and K. FLICK. Deutsche Ztschr. f. Chir., 1926, cxcviii, 63.

The diagnosis and practical treatment of important dislocations and fractures involving joints. R. BONN. Beihefte z. Med. Klin., 1926, xxii, 991.

Osteosynthesis and compound fractures. H. GAUDIER and E. GAUDIER. Bull. et mém. Soc. nat. de chir., 1926, lii, 877.

Osteosynthesis with a buried prosthesis in cases of complicated diaphyseal fractures. R. SIMON, E. STULZ, and C. LENORMANT. Bull. et mém. Soc. nat. de chir., 1926, lii, 562.

The cause and treatment of ununited fractures. M. S. HENDERSON. South. M. J., 1926, xix, 746. **[322]**

Snapping shoulder. BRANDT. Zentralbl. f. Chir., 1926, liii, 2550.

Recurring luxation of the shoulder. OUDARD. Bull. et mém. Soc. nat. de chir., 1926, lii, 882.

The treatment of habitual luxation of the shoulder. E. KOENIG. Zentralbl. f. Chir., 1926, liii, 1174.

The results of operations for habitual dislocation of the shoulder, with special consideration of our method. G. PERTHES. Deutsche Ztschr. f. Chir., 1925, cxciv, 1.

A simplified operative procedure for habitual dislocation of the shoulder. F. RUPP. Deutsche Ztschr. f. Chir., 1926, cxcviii, 70.

Open reduction of an old fracture of the shoulder. AUVRAY. Bull. et mém. Soc. nat. de chir., 1926, lii, 911.

Dislocation of the outer end of the clavicle. A. B. MITCHELL. Brit. M. J., 1926, ii, 1097.

Two cases of total luxation of the clavicle. F. BOEHM. Deutsche Ztschr. f. Chir., 1926, cxcvi, 307.

Adjustable extension apparatus for the upper extremity. R. FRANK. Zentralbl. f. Chir., 1926, liii, 2206.

Intentional fracture of the humerus in the newborn. D. F. CLARK. Surg., Gynec. & Obst., 1926, xliii, 795.

Fractures of the humerus and radial paralysis. J. L. ROUX-BERGER. Bull. et mém. Soc. nat. de chir., 1926, lii, 551.

The mechanism and treatment of backfire fracture. H. C. EDWARDS. J. Bone & Joint Surg., 1926, viii, 701. **[322]**

Fracture of the neck of the radius with displacement of the fragments; fracture of the ulna in the upper third; osteosynthesis. GRIMAULT and MOUCHET. Bull. et mém. Soc. nat. de chir., 1926, lii, 893.

The treatment of luxation of the semilunar bone. A. WITTEK. Wien. klin. Wchnschr., 1926, xxxix, 1097.

Subtotal retrolunar luxation of the left carpus with fracture of the cuneiform: early reduction; recovery. SOLCARD, COUREAUD, and MOUCHET. Bull. et mém. Soc. nat. de chir., 1926, lii, 891.

Fractures of the transverse processes of the vertebræ. M. SCHIESSL. Deutsche Ztschr. f. Chir., 1926, cxcvi, 297.

Laminectomy in a case of complete luxation fracture in the cervical portion of the spinal column. K. HAUGSETH. Norsk Mag. f. Laegevidensk., 1926, lxxxvii, 298.

Compression fracture of the lumbar vertebræ from slight trauma. W. MULL. Deutsche Ztschr. f. Chir., 1926, cxcvi, 291.

End-results of vertebral fractures. HAUMANN. 50 Tag. d. deutsch. Ges. f. Chir., Berlin, 1926.

A case of luxation of the right half of the pelvis. S. DAVIDOVIC. Zentralbl. f. Chir., 1926, liii, 2203.

Two direct symptoms of luxation of the hip. A. BÁRON. Zentralbl. f. Chir., 1926, liii, 2457.

The open palliative treatment of dislocation of the hip from a mechanical standpoint. A. FARKAS. Ztschr. f. orthop. Chir., 1926, xlvii, 356, 364.

A case of spontaneous fracture of the hip. P. D. WILSON. Boston M. & S. J., 1926, cxcv, 1205.

Fracture at the site of an old osteomyelitis; fracture at the lower end of the femur. C. A. HULL. Nebraska State M. J., 1926, xi, 479.

Two cases of subtrochanteric fracture of the femur. PLISSON. Bull. et mém. Soc. nat. de chir., 1926, lii, 912.

The treatment of fractures of the femur. E. KEY. Zentralbl. f. Chir., 1926, liii, 1129.

Lateral dislocation of the patella complicated by rotation. E. ORSÓS. Zentralbl. f. Chir., 1926, liii, 1506.

Traumatic luxation of the knee. R. ZANOLI. Chir. d. organi di movimento, 1926, xi, 73.

A light portable extension frame. W. A. CLARK. J. Bone & Joint Surg., 1926, viii, 750. **[323]**

A traction splint for the leg. R. FRANK. Zentralbl. f. Chir., 1926, liii, 2343.

A new immobilization extension apparatus for the lower extremities. A. BRAUN. Muenchen. med. Wchnschr., 1926, lxxiii, 1115.

A case of fracture of the base of the fifth metatarsal due to muscular action. O. MAGATON. Chir. d. organi di movimento, 1926, xi, 94.

An unusual type of fracture of the os calcis. E. OPAZO. Bol. Soc. de cirug. de Chile, 1926, iv, 159.

Os calcis fractures: a new method of reduction. M. C. HARDING. J. Bone & Joint Surg., 1926, viii, 720.

Orthopedics in General

Autoplasty and alloplasty in orthopedics. F. LANGE. Ztschr. f. orthop. Chir., 1926, xlvii, 211, 269.

Working toward the correction of Kentucky's crippled children. M. WILLIAMSON. Kentucky M. J., 1926, xxiv, 619.

A textbook of massage. J. H. LUBINUS. 1926: Munich, Bergmann.

A simple wrist exerciser. W. K. MORRISON. J. Roy. Army Med. Corps, Lond., 1926, xlvii, 445.

Crutch-walking as an art. W. G. WRIGHT. Am. J. Surg., 1926, n.s. i, 372.

Orthopedic problems in leprosy. P. A. MCILHENNY. J. Am. M. Ass., 1926, lxxxvii, 1888.

Investigations concerning the photo-activity of certain oils with special reference to their effect as antirachitics. E. REKLING. Acta radiol., 1926, v, 517.

SURGERY OF THE BLOOD AND LYMPH SYSTEMS

Blood Vessels

An interesting case of aneurism from the clinical and operative standpoint. H. HABERER. Deutsche Ztschr. f. Chir., 1926, cxcvi, 215.

Aneurism of the subclavian artery; ligation of the first division and intrasaccular suture. S. C. G. KIRCHNER. Surg., Gynec. & Obst., 1926, xliii, 764.

A case of genuine diffuse varicose aneurism of the upper arm. S. ERDHEIM. Wien. med. Wchnschr., 1926, lxxvi, 603.

Arteriovenous aneurism between the internal carotid and the cavernous sinus. A. CROCCO. Semana méd., 1926, xxxiii, 1075.

A case of multiple aneurisms of the branches of the pulmonary artery. D. H. SHERMAN and B. ROMAN. Bull. Buffalo Gen. Hosp., Buffalo, N. York, 1926, iv, 39.

Hernia of the superior epigastric artery. J. H. MORRIS. Surg., Gynec. & Obst., 1926, xliii, 767.

Aneurism of the right hypogastric artery simulating a tumor of inflammatory nature. BRUNNER. Clin. Ostet., 1926, xxviii, 498.

Aneurism of the renal artery. W. P. CALLAHAN and F. H. SCHILTZ. Surg., Gynec. & Obst., 1926, xliii, 724.

Traumatic arterial aneurism of the base of Scarpa's triangle. H. GROVE, C. SOROLLA, A. SCHOENHERR, and ALVAREZ. Arch. Soc. méd. de Valparaíso, 1926, i, 74.

Aneurisms of the popliteal and external iliac artery. F. OPAZO. Bol. Soc. de cirug. de Chile, 1926, iv, 151.

A case of saccular aneurism of the posterior tibial artery: complete exposure and extirpation of the sac. H. C. SIDGWICK. J. Roy. Army Med. Corps, Lond., 1926, xlvii, 448.

Fascial bands in the treatment of aneurism. J. L. CAMPBELL. South. M. J., 1926, xix, 795.

Angiostomy. H. F. O. HABERLAND. Ztschr. f. d. ges. exper. Med., 1926, l, 180.

Surgical experiences with regard to the carotid sinus blood pressure. M. BUDDE. Ztschr. f. d. ges. exper. Med., 1926, l, 207.

Primary ligation of the external carotid artery. H. HERFARTH. Zentralbl. f. Chir., 1926, liii, 1106.

Anomalies of the subclavian artery. R. BELLEVILLE and G. I. BELLEVILLE. Rev. de cirug., Buenos Aires, 1926, v, 478.

Resection of a portion of the axillary artery. F. W. CELCE. Med. J. & Rec., 1926, cxxiv, 697.

The surgical anatomy of the bifurcation of the aorta. A. J. SSOSON-JAROSCHEWITSCH. Ztschr. f. d. ges. Anat.,

Part I. Ztschr. f. Anat. u. Entwicklungsgesch., 1926, lxxix, 44.

A stab wound of the abdominal aorta. WILDEGANS. Zentralbl. f. Chir., 1926, liii, 2281.

Sarcomatous permeation of the inferior vena cava and the right side of the heart. H. J. B. FRY and C. E. SHATTOCK. Brit. J. Surg., 1926, xiv, 337. [324]

The question of so-called phlebitis migrans. B. HERZBERG. Beitr. z. klin. Chir., 1926, cxxxvii, 29.

Thrombophlebitis of the utero-ovarian vein. INFANTOZZI. Rev. méd. d. Uruguay, 1926, xxix, 235.

Embolectomy. BONNET and JENTZER. Lyon chir., 1926, xxiii, 618.

The prognosis and the technique of embolectomy. G. NYSTROEM. Zentralbl. f. Chir., 1926, liii, 1131.

Sarcoma of the sheath of the femoral vessels. CHALIER. Lyon chir., 1926, xxiii, 587.

Blood; Transfusion

Is the blood a mirror of the constitution? H. SELLHEIM. Karlsbader aerztl. Vortr., 1926, vii, 78.

A new needle for withdrawing blood. L. A. BARINSTEIN. Zentralbl. f. Chir., 1926, liii, 1180.

The form in which calcium exists in the serum. A. NITSCHKE and H. J. FREYSCHMIDT. Biochem. Ztschr., 1926, clxxiv, 287.

The effect of calcium lactate ingestion on the serum calcium. W. BAUER and M. W. ROPES. J. Am. M. Ass., 1926, lxxxvii, 1902.

Studies in blood glycolysis: general consideration of glycolysis in relation to the blood cells and the production of lactic acid and carbon dioxide. I. KATAYAMA. J. Lab. & Clin. Med., 1926, xii, 239.

A comparison of the Folin-Wu and new Benedict methods for the estimation of blood sugar. A. E. OSTERBERG and J. STRUNK. J. Lab. & Clin. Med., 1926, xii, 278.

Postmortem blood-sugar determinations. J. R. PAUL. Bull. Ayer Clin. Lab. Pennsylvania Hosp., 1926, No. 10, 34.

The influence of roentgen radiation on cholesterin content of the blood in normal and pathological conditions. LEVY-DORN and BURGHEIM. Fortschr. a. d. Geb. d. Roentgenstrahlen, 1926, xxxiv, 62.

Bilirubinæmia considered especially as a symptom of appendicitis and of extravasations of blood (icterus of the newborn). FRIDTJOF BANG. Gynéc. et obst., 1926, xiv, 223.

Volume measurement of blood platelets. C. M. VAN ALLEN. J. Lab. & Clin. Med., 1926, xii, 282.

The clinical importance of the blood sedimentation reaction: experiences in more than 3,000 cases. G. HASELHORST. Deutsche med. Wchnschr., 1926, lii, 1544.

Some observations upon erythrocyte sedimentation. FIGUERIDO and BILBAO. Arch. de med., cirug. y especial., 1926, vii, 56.

The effect of roentgen radiation on the red blood cells. F. KROEMEKE. Fortschr. a. d. Geb. d. Roentgenstrahlen, 1926, xxxiv, 63.

The roentgen treatment of leukæmias. E. L. JENKINSON. Radiology, 1926, vii, 379.

A case of acute leukæmia. C. SCHWARZENBERG. Arch. Soc. méd. de Valparaíso, 1926, i, 80.

A case of lymphatic leukæmia of unusual type. Ä. E. NAISH and C. TINGLE. Lancet, 1926, ccxi, 1156.

Recurring epistaxis from chronic hæmorrhagic purpura. W. J. MAYO and A. C. JOHNSON. Surg. Clin. N. Am., 1926, vi, 1121.

Two cases of purpura hæmorrhagica. C. J. FONSECA. Repert. de med. y cirug., 1926, xvii, 674.

Essential thrombocytopænia (purpura hæmorrhagica). A. B. HODGES. Virginia M. Month., 1926, liii, 582.

Dyscrasias of the blood. W. J. MAYO. New Orleans M. & S. J., 1926, lxxix, 299. [324]

The surgical aspect of blood dyscrasias. J. SPEESE. Ann. Surg., 1926, lxxxiv, 477. [325]

The technique of blood group determination for clinicians and medicolegal experiences with particular reference to its use in anthropology and in the study of heredity and constitution. F. SCHIFF. 1926: Berlin, Springer.

A case of transfusion of incompatible blood: the tests of compatibility. P. MORVILLE. Bruxelles-méd., 1926, vi, 1562.

Practical aspects of transfusion. E. I. SPRIGGS. Brit. M. J., 1926, ii, 978.

Indications for blood transfusion. SIR H. ROLLESTON. Brit. M. J., 1926, ii, 969. [325]

Transfusion of blood from animal to man. R. CRUCHET. Brit. M. J., 1926, ii, 975. [325]

Blood transfusion in surgery. G. KEYNES. Brit. M. J., 1926, ii, 980. [325]

Blood transfusions, indications and methods. A. A. STRAUSS. J. Michigan State M. Soc., 1926, xxv, 623.

Pernicious anæmia and blood transfusion. G. L. GULLAND. Brit. M. J., 1926, ii, 973.

Intraperitoneal transfusion of citrated blood in acute intestinal intoxications of infancy. C. F. MCKHANN. Boston M. & S. J., 1926, cxcv, 1241.

Intraperitoneal transfusion in von Jaksch's anæmia. C. G. GRULEE. N. York State J. M., 1926, xxvi, 921. [326]

Fatal acute nephritis following a blood transfusion. SANTY. Lyon chir., 1926, xxiii, 608.

Lymph Vessels and Glands

The lymphatic system as a defense mechanism. C. W. M. POYNETER. Colorado Med., 1926, xxiii, 386.

Lymphangioma and its surgical treatment. B. BRANDSBURG. Arch. f. klin. Chir., 1926, cxli, 377.

Two cases of lymphogranulomatosis of the groin. VERDELET and AUCHÉ. J. de méd. de Bordeaux, 1926, ciii, 739.

SURGICAL TECHNIQUE

Operative Surgery and Technique; Postoperative Treatment

Surgery and organ function. E. REHN. 50 Tag. d. deutsch. Ges. f. Chir., Berlin, 1926. [327]

The importance of a careful study of the personality in deciding the indication for operation, particularly in women. H. CABOT. Internat. J. Med. & Surg., 1926, xxxix, 485.

The prognosis in operative surgery. A. FRAENKEL. Wien. klin. Wchnschr., 1926, xxxix, 1061.

Some observations on the immediate prognosis of major operations. O. C. CASSEGRAIN. New Orleans M. & S. J., 1926, lxxix, 396.

Pre-operative and postoperative treatment. D. S. CUNEO. Rev. de cirug., Buenos Aires, 1926, v, 450.

Life-saving details. F. B. SWEET. Boston M. & S. J., 1926, cxcv, 1233.

Diabetes, insulin, and surgery. A. W. FISCHER. Ergebn. d. Chir. u. Orthop., 1926, xix, 1.

The use of insulin in surgery. J. A. NIXON. Bristol M.-Chir. J., 1926, xliii, 199.

Pre-operative study in cases of renal insufficiency. C. K. SMITH. Med. Herald & Physiotherap., 1926, xlv, 319.

The treatment of cutaneous burns. F. W. BANCROFT, A. O. WHIPPLE, S. ERDMAN, and others. Ann. Surg., 1926, lxxxiv, 892.

The treatment of old roentgen ulceration. VON TEMPSKY. Zentralbl. f. Chir., 1926, liii, 1218.

Plastic operation with skin flaps displaced in a wavelike manner. PILZ. Zentralbl. f. Chir., 1926, liii, 2282.

Celluloid as an alloplastic material in animal experimentation. C. E. JANCKE. Deutsche Ztschr. f. Chir., 1926, cxcvii, 18.

Experiences with stryphnon as a coagulative medium. E. LIHOTZKY. Zentralbl. f. Chir., 1926, liii, 2342.

Resuscitation by intracardiac injection of adrenalin. R. CHALMERS. Brit. M. J., 1926, ii, 1223.

Paralysis of the respiratory center lasting for hours with restoration of function. H. SELLHEIM. Med. Klin., 1926, xxii, 1323.

Recovery after massage of the heart. L. COOK. Brit. M. J., 1926, ii, 1118.

Further contributions on failures and mistakes in surgery. C. PRIMA. Zentralbl. f. Chir., 1926, liii, 1637.

The etiology, prevention, and treatment of postoperative wound infections. M. THOREK. Illinois M. J., 1926, l, 477.

The question of postoperative non-diabetic acidosis. M. HERZ. Zentralbl. f. Chir., 1926, liii, 1684.

Pulmonary postoperative complications. L. TORRACA. Ann. ital. di chir., 1926, v, 853.

More recent studies upon the etiology of postoperative pneumonitis. A. O. WHIPPLE. N. York State J. M., 1926, xxvi, 967.

Antiseptic Surgery; Treatment of Wounds and Infections

A new German Surgery: H. KUETTNER. Vol. XX: A handbook of wound treatment: C. BRUNNER. 1926: Stuttgart, Enke.

Wound treatment in medical practice. BERKENBUSCH. Muenchen. med. Wchnschr. 1926, lxxiii, 570.

Disinfection of fresh wounds. H. L. CHAMBERS. J. Kansas M. Soc., 1926, xxvi, 383.

Essential immediate treatment of trauma. H. C. W. S. DE BRUN. Am. J. Surg., 1926, n.s. i, 376.

Physical measures in acute trauma. M. A. COHEN. Am. J. Surg., 1926, n.s. i, 386.

Local immunity in surgical practice. D. DE FRANCESCO. Ann. ital. di chir., 1926, v, 709.

Acid therapy in minor surgery. A. JALCOWITZ and W. SCHOSSERER. Wien. klin. Wchnschr., 1926, xxxix, 989.

Protein therapy, specific and non-specific. A. SCHNEIDER. Am. Med., 1926, xxxii, 753.

Autohæmotherapy. L. GOLDEMBERG. Semana méd., 1926, xxxiii, 1025.

Remarks on chinosol. W. HEUBNER and R. SIEGEL. Klin. Wchnschr., 1926, v, 1709.

A case of injury due to a penpoint. R. GARTENMEISTER. Zentralbl. f. Chir., 1926, liii, 1687.

Penpoint injuries and their treatment. G. MAGNUS. Fortschr. d. Therap., 1926, ii, 383.

The static current in the treatment of inflammation. W. B. SNOW. Med. J. & Rec., 1926, cxxiv, 769.

The roentgen-ray treatment of subacute infections. V. W. ARCHER. Virginia M. Month., 1927, liii, 649.

The roentgen ray in the treatment of certain localized infections. F. M. HODGES. South. M. J., 1926, xix, 857.

The origin and evolution of putrid infections and their treatment by serum; experimental studies. WEINBERG and GINSBOURG. Arch. de med., cirug. y especial., 1926, vii, 5.

The closed cavity procedure of Méndez in the treatment of infections with purulent or puriform secretions. L. G. GRET. Semana méd., 1926, xxxiii, 1071.

Streptococcic serotherapy and the blood picture, with an investigation of the objective value of serotherapy in sepsis. H. LEHFELDT. Zentralbl. f. Gynaek., 1926, l, 1564.

Anæsthesia

The ideal anæsthetic. F. H. MCMECHAN. Brit. M. J., 1926, ii, 1106.

Some physical factors in the administration of gaseous anæsthetics. E. I. MCKESSON. Brit. M. J., 1926, ii, 1113.

Concerning the intermediate stage in anæsthesia. E. VON D. PORTEN. Zentralbl. f. Chir., 1926, liii, 1682.

The use of somnifene, pantopon, and atrinal for general anæsthesia. M. HAEGI and C. HUYSSEN. Schweiz. med. Wchnschr., 1926, lvi, 531.

Intravenous anæsthesia with somnifene. G. DUETTMANN. Med. Klin., 1926, xxii, 1419.

Positive pressure narcosis with narcylen. H. KILLIAN. Arch. f. klin. Chir., 1926, cxli, 357.

A contribution upon the effect of narcylen anæsthesia upon the blood pressure and the nervous system. H. FRANKEN, H. BOELLERT, and O. EICHLER. Zentralbl. f. Gynaek., 1926, l, 2461.

The causes of increased bleeding in operations performed under narcylen anæsthesia. HILDEBRANDT, BOELLERT, and EICHLER. Klin. Wchnschr., 1926, v, 1756.

The advantages and disadvantages of anæsthesia with ether, narcylen, and nitrous oxide. P. SUDECK. Beitr. z. klin. Chir., 1926, cxxxvii, 493.

Possible after-effects of narcylen anæsthesia. E. SEIFERT. Muenchen. med. Wchnschr., 1926, lxxiii, 559.

The production and preservation of sterile solutions, with particular reference to tutocain. H. STEICHELE. Ztschr. f. d. ges. Krankenhauswes., 1926, xxii, 524.

Tutocain and dolantin. H. LANGSDORFF. Deutsche Monatsschr. f. Zahnh., 1926, xliv, 371.

Spinal anæsthesia. J. IZQUIRDO. Deutsche med. Wchnschr., 1926, lii, 646.

One hundred and eighteen cases of spinal anæsthesia. C. A. CASTRO. Semana méd., 1926, xxxiii, 1158.

Spinal anæsthesia with tutocain. L. L. PEREIRA. Rev. med.-quirurg. hospitales, 1926, ii, 347.

Lumbar anæsthesia in surgery. J. ARCE. Deutsche med. Wchnschr., 1926, lii, 644.

Conduction anæsthesia of the lumbar plexus. H. DEGENHARDT. Zentralbl. f. Chir., 1926, liii, 1570.

Vasomotor collapse in lumbar anæsthesia and its prevention. H. ZIEGNER. Zentralbl. f. Chir., 1926, liii, 1367.

Modification of local anæsthesia with an alkaloid of quinine. VON TAKÁTS and T. PAUNZ. Klin. Wchnschr., 1926, v, 1324.

Cibalgin as an analgesic in surgery. A. LOEWENTHAL. Deutsche med. Wchnschr., 1926, lii, 963.

PHYSICOCHEMICAL METHODS IN SURGERY

Roentgenology

A charging device for the electroscope. R. B. TAFT. Radiology, 1926, vii, 513.

Roentgenology in Russia. I. S. HIRSCH. Radiology, 1926, vii, 463.

Proposition for a portable orthodiagraph. L. BISCHOFF. Brit. J. Radiol., 1926, xxxi, 510.

Some brominized oils for radiographic use: preliminary report. T. J. PUTNAM. J. Am. M. Ass., 1926, lxxxvii, 1102. [329]

The measurement of quantitative biological effects of the X-rays. C. PACKARD. J. Cancer Research, 1926, x, 319.

A simple and reliable rule for the measurment of deep dosage. R. M. SIEVERT. Acta radiol., 1926, v, 468.

Stimulation and immunity in radiotherapy. A. U. DESJARDINS. J. Am. M. Ass., 1926, lxxxvii, 1537. [329]

Hyperæmia as a sensitization to secondary rays in roentgen radiation. W. WYNEN. Strahlentherapie, 1926, xxii, 503.

Experiments on the selectivity of the secondary radiations. M. PONZIO. Brit. J. Radiol., 1926, xxxi, 502.

Deep X-ray therapy. I. GERBER and S. ALBERT. Rhode Island M. J., 1926, ix, 185.

The effect of radiation on the Rous chicken tumor. J. C. MOTTRAM. Lancet, 1926, ccxi, 1266.

Radio-active material an industrial hazard? F. B. FLINN. J. Am. M. Ass., 1926, lxxxvii, 2078.

On photographic marking of roentgen negatives. P. HAGLUND. Acta radiol., 1926, v, 471.

Radium

The physics and chemistry of radium and mesothorium for physicians and students. A. FERNAU. 1926: Vienna, Springer.

Radon (radium emanation) as a palliative agent in the treatment of intra-oral cancer. F. E. SIMPSON and R. E. FLESHER. J. Cancer, 1926, iii, No. 12, 18.

Miscellaneous

What is physical therapy? N. E. TITUS. Med. Hosp., 1926, xxvii, 54.

Some high lights on physiotherapy. F. W. WILLIS. J. Nat. M. Ass., 1926, xviii, 204.

Physiotherapy: its use by the general practitioner. J. W. SEVER. Boston M. & S. J., 1926, cxcv, 1099.

Physiotherapy in surgery. G. C. COOKE. South. M. & S., 1926, lxxxviii, 795.

Indications for actinotherapy. V. PEÑA. Rev. de med. y cirug., Caracas, 1926, ix, 130.

An increase of ferment in the serum following light and roentgen-ray irradiation, an index of the processes of cell destruction. A. MERTZ. Strahlentherapie, 1926, xxii, 301. [329]

Sunlight as a disinfectant. F. M. MEADER. J. Michigan State M. Soc., 1926, xxv, 636.

Heliotherapy and actinotherapy in relation to pediatrics. F. W. SCHLUTZ. Am. J. Dis. Child., 1926, xxxii, 900.

From heliotherapy to heliohygiene. C. W. SALEEBY. Med. Press., 1926, n.s. cxxii, 443.

Ultraviolet light a fundamental necessity in therapeutics. I. W. JENKINS. Texas State J. M., 1926, xxii, 510.

The use and misuse of ultraviolet therapy. A. F. HESS. N. York State J. M., 1926, xxvi, 816. [330]

Ultraviolet radiation: some illustrative cases. A. L. PUNCH and R. WILKINSON. Lancet, 1926, ccxi, 1211.

Multothermy: the new science of the application of electric energy to medicine and surgery. H. GOODMAN. Med. Herald & Physiotherap., 1926, xlv, 326.

Medical and surgical diathermy. W. D. MCFEE. Internat. J. Med. & Surg., 1926, xxxix, 481.

The double electrode and diathermy instruments. L. SAMENGO. Semana méd., 1926, xxxiii, 1113.

The treatment of moist gangrene in diabetics by diathermy. D. PAÏZIS. Presse méd., Par., 1926, xxxiv, 1242. [330]

Use of diathermy in external new growths outside mucous cavities. R. VALLACK. Med. J. Australia, 1926, ii, 764.

MISCELLANEOUS

Clinical Entities—General Physiological Conditions

Familial situs inversus. A. H. HOFMANN. Zentralbl. f. Chir., 1926, liii, 1633.

Hemihypertrophy. L. H. MAYERS. Surg., Gynec. & Obst., 1926, xliii, 746.

Diabetes insipidus with polyuria of 40 liters. J. C. NAVARRO. Semana méd., 1926, xxxiii, 1125.

The surgical problem in diabetes. F. PICCALUGA. Rev. méd. Lat.-Am., 1926, xii, 70.

Raynaud's disease—a pathological entity—report of a case following the Leriche operation. W. F. CUNNINGHAM. N. York State J. M., 1926, xxvi, 972.

The reticulo-endothelial apparatus in burns. C. CARACCÒ. Ann. ital. di chir., 1926, v, 925.

Industrial and domestic electrical accidents. A. ZIMMERN. Bruxelles-méd., 1926, vi, 1461.

An electrical burn. TAVERNIER and GELAS. Lyon chir., 1926, xxiii, 626.

Leg ulcers. J. W. BARR. Atlantic M. J., 1926, xxx, 156.

The metabolism of tumors. O. WARBURG. 1926: Berlin, Springer.

Studies on experimental tumors. M. LAPIDARI. Riforma med., 1926, xlii, 891.

Distribution of arsenic in tumor-bearing mice. A. S. MINOT. J. Cancer Research, 1926, x, 293.

The influence of insufficient diets upon tumor recurrence and growth in rats and mice. K. SUGIURA and S. R. BENEDICT. J. Cancer Research, 1926, x, 309.

Conjunctivoma of the soft parts of the thigh. TIXIER. Lyon chir., 1926, xxiii, 589.

Fibroma of the sole of the foot. L. H. PETIT. Bull. et mém. Soc. nat. de chir., 1926, lii, 876.

Study of malignant disease in the State Institute, Buffalo, N. Y. B. T. SIMPSON. N. Y. State J. M., 1926, xxvi, 979.

Cancer and public health. A. CHARLES. J. Cancer, 1926, iii, No. 11, 24.

Cancer mortality in the Argentine. A. H. ROFFO. Cancer, 1926, iii, 55.

The causes of carcinoma. R. P. VAN CALCAR. 1926: Leyden, Van Doesburgh.

The etiology of cancer. L. D. BRISTOL. Med. J. & Rec., 1926, cxxiv, 747.

New research in the etiology of cancer, with reference to the work of Gye. M. J. SITTENFIELD. Am. J. Roentgenol., 1926, xvi, 525.

Pathological currents and cancer. H. BYRD. J. Cancer, 1926, iii, No. 12, 13.

The nerve factor in relation to cancer. C. HICKEY. J. Cancer, 1926, iii, No. 12, 1.

Cancer and neuronal control. H. BROWN. J. Cancer, 1926, iii, No. 12, 22.

Malignant melanotic tumors in the negro. J. T. BAUER. Bull. Ayer Clin. Lab. Pennsylvania Hosp., 1926, No. 10, 5.

Tar cancer in the white mouse: an experimental study. G. DOEDERLEIN. Ztschr. f. Krebsforsch., 1926, xxiii, 241.

Superficial epitheliomata. C. J. LUNSFORD and L. TAUSSIG. California & West. Med., 1926, xxv, 740.

The transplantation of human carcinoma. H. KURTZAHN. Klin. Wchnschr., 1926, v, 1166.

Abnormal cancer metastases following lead treatment. E. E. GLYNN, R. KENNON, H. S. PEMBERTON, and C. O. DAVIES. Brit. M. J., 1926, ii, 1224.

The cure of cancer by its early recognition and its prevention. R. MILNER. 1926: Leipzig, Hirzel.

Malignant disease and a biological immunization therapy. W. SCHMIDT. J. Cancer, 1926, iii, No. 11, 1.

General Bacterial, Protozoan, and Parasitic Infections

Sepsis from the pathologico-anatomical standpoint. KAISERLING. Deutsche med. Wchnschr., 1926, lii, 1199. [331]

The localization in animals of bacteria isolated from foci of infection. A. C. NICKEL. J. Am. M. Ass., 1926, lxxxvii, 1117. [331]

Deep phlegmon of the thigh due to diphtheria. J. ABADIE. Bull. et mém. Soc. nat. de chir., 1926, lii, 868.

Tetanus. W. G. KEMPER. J.-Lancet, 1926, xlvi, 572.

Oxygen in gas bacillus infections. A. J. HOSMER. Am. J. Surg., 1926, n.s. i, 391.

Clinical and experimental studies of the treatment of localized anthrax infection by localizing injections of blood according to Laewen's method. K. H. ERB. Beitr. z. klin. Chir., 1926, cxxxvii, 202.

Tissue-digesting enzyme (histase) of streptococci. M. FROBISHER, JR. J. Exper. Med., 1926, xliv, 777.

The treatment of typhoid and paratyphoid fever with staphylococcic vaccine. V. BIE. Acta med. Scand., 1926, lxv, 160.

Notes on a case of meningococcal septicæmia. H. T. FINDLAY and R. N. PHEASE. J. Roy. Army Med. Corps, Lond., 1926, xlvii, 445.

Acute nodular leprosy originating in this country and cured by vaccine treatment. E. G. LITTLE. Brit. M. J., 1926, ii, 1034.

Hydatid disease in man. FULTON. N. Zealand M. J., 1926, xxv, 317.

Laboratory aids in the diagnosis of hydatid disease. HERCUS. N. Zealand M. J., 1926, xxv, 323.

A case of generalized amœbiosis. G. FRANCHINI. Riforma med., 1926, xlii, 843.

Tularæmia, with report of 11 cases. B. C. FARRAND. Am. J. M. Sc., 1926, clxxii, 853.

Human blastocystis; its pathological significance and frequency in Buenos Aires. S. E. PARODI and F. L. NIÑO. Semana méd., 1926, xxxiii, 1058.

Surgical treatment of certain massive blastomycotic skin lesions. R. H. JACKSON. Am. J. Surg., 1926, n.s. i, 185. [331]

Ductless Glands

Relation of the gonads to the other glands of internal secretion. H. L. HUNT. Med. Times, 1926, liv, 293.

The dependence of disturbances of growth and bone diseases on disturbances of internal secretion. O. MAAS. Samml. zwangl. Abhandl. a. d. Geb. d. Verdauungs- u. Stoffwechs.-Krankh., 1926, ix, 1.

An endocrine family. J. H. HUTTON. Illinois M. J., 1926, l, 473.

The diagnosis and treatment of certain glandular deficiencies. L. G. ROWNTREE and A. M. SNELL. Med. Clin. N. Am., 1926, x, 513. [332]

Remarks on hormone therapy. H. HANDOVSKY. Deutsche med. Wchnschr., 1926, lii, 823.

Surgical Pathology and Diagnosis

The chemistry of cell staining. VIII. The recognition of lipoids in cells and tissue. J. SCHUMACHER. Chemie d. Zelle u. Gewebe, 1926, xii, 433.

Studies of the processes of swelling and pathological changes in the connective tissue. A. DIETRICH. Zentralbl. f. allg. Path. u. path. Anat., 1926, xxxvii, 156.

The disintegrating of tissue. W. VON GAZA. Klin. Wchnschr., 1926, v, 1072.

The differential diagnosis of exudates and transudates. P. CORSONELLO. Riforma med., 1926, xlii, 844.

Celluloid jars for mounting permanent specimens. D. H. DOUGLAS. J. Obst. & Gynæc. Brit. Emp., 1926, xxxiii, 660.

Experimental Surgery

The operative technique of animal experimentation. H. F. O. HABERLAND. 1926: Berlin, Springer.

Some effects of tar applied paracutaneously. V. E. MERTENS. Ztschr. f. Krebsforsch., 1926, xxiii, 351.

Hospitals; Medical Education and History

Medical education and its product. W. H. MOURSUND. South. M. J., 1926, xix, 896.

The importance of the library in medical practice. W. S. THAYER. Med. J. & Rec., 1926, cxxiv, 715.

The service of the (New York) Academy. S. A. BROWN. Med. J. & Rec., 1926, cxxiv, 721.

The men who made the (New York) Academy of Medicine. L. R. WILLIAMS. Med. J. & Rec., 1926, cxxiv, 722.

The needs and future of the (New York) Academy Library. A. MALLOCH. Med. J. & Rec., 1926, cxxiv, 723.

The librarian of the (New York) Academy of Medicine. D. B. DELAVAN. Med. J. & Rec., 1926, cxxiv, 725.

The library of the New York Academy of Medicine J. S. BROWNNE. Med. J. & Rec., 1926, cxxiv, 726.

SUPPLEMENT I

(KOLODNY: *Bone Sarcoma*)

A STUDY OF THE MATERIAL FROM THE REGISTRY OF BONE SARCOMA OF THE AMERICAN COLLEGE OF SURGEONS

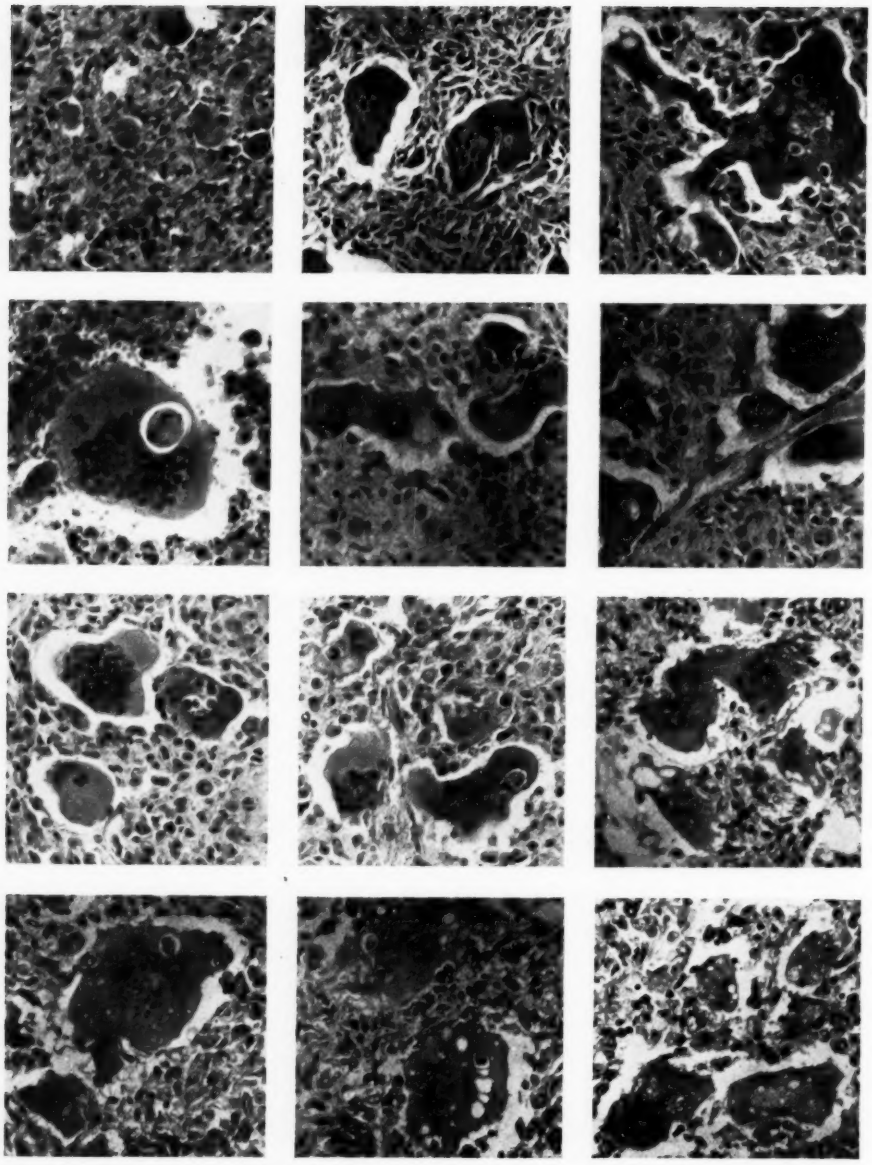


Plate 37. Composed of photomicrographs of cases of giant cell tumor of the Registry collection. Showing vagaries of giant cells of the epulis type. Formation and degeneration of giant cells.

BONE SARCOMA

THE PRIMARY MALIGNANT TUMORS OF BONE AND THE GIANT CELL TUMOR

BY

ANATOLE KOLODNY, Ph.D., M.D.

DEPARTMENT OF SURGERY
STATE UNIVERSITY OF IOWA

AUTHORIZED BY THE AMERICAN COLLEGE OF SURGEONS
THE SURGICAL PUBLISHING COMPANY OF CHICAGO, PUBLISHER
1927

Copyright by
The Surgical Publishing Company
of Chicago
1927

RESPECTFULLY DEDICATED

TO

THE ORGANIZER OF THE REGISTRY OF

BONE SARCOMA OF THE AMERICAN COLLEGE OF SURGEONS

AND THE FIRST REGISTRAR

ERNEST AMORY CODMAN, M.D.

Whose tireless work and

idealism has inspired a generation of students

in their

investigation of bone tumors.

"Where facts are numerous and unquestionable, and unequivocal in their significance, theory must follow them as it best may, keeping time with their step, and not go before them, marching to the sound of its own drum and trumpet."

OLIVER WENDELL HOLMES, 1842

PREFACE

THE experience of the Registry of Bone Sarcoma of the American College of Surgeons brought much light in the dark field of malignant bone tumors. For various reasons, however, few clinicians and laboratory men have had the opportunity to study the material and to share in the gains of the Registry. It seemed appropriate at this point to assemble, analyze and critically present before the profession at large the multitude of facts and information gained from a personal study of the 700 cases of the Registry and an extensive review of the literature to date. In consequence of these considerations I attempted to facilitate the comprehension of the frequently opposing opinions by presentation of the views at which I arrived after repeated studying of this material. I court no delusion as to the brevity and inadequacy of the discussion of some questions in this treatise; my desire to present only basic and essential facts as gained from the study of the Registry collection is my excuse. My primary aim is to assist the diffusion of the experience of the Registry as it stands today. I entirely agree with Codman when he says that the opinion of one who has studied "this series of cases could certainly be of help to anyone on whom the responsibility of decision of life and limb rests."

In the chapter on classification I am presenting a critical analysis of the classification offered by the Registry Committee and am suggesting certain changes in it. In the chapter on osteogenic sarcoma I have discussed at considerable length the general principles of the pathological diagnosis and prognosis common to all primary malignant bone tumors. Because of the intimate association of the therapy and prognosis of bone sarcoma these two must needs overlap, so that in osteogenic sarcoma both these chapters are best studied together; in the other tumors both these questions are considered in a single chapter.

While the recognition of the benignity of giant cell tumor has had a clarifying influence and while the description of its pathology has contributed much to a better insight into the concept of this disease, the relation of it to malignant bone tumor is still enshrouded in mystery in the minds of the average clinician and pathologist. Multiplicity of causal modalities brings forth identical phenomena and by virtue of overlapping manifestations of varied origin intricate clinical complexes result. Thus lesions of varied motivation must needs be considered in connection with and in relation to malignant tumors that may bring about a similar diagnostic and prognostic issue. I have therefore included in this mono-

graph the giant cell tumor that occasionally calls forth clinical symptoms similar to malignant bone sarcoma.

The illustrations are selected from the material of the Registry. Only a few roentgenograms are brought in of cases observed by me elsewhere. Not all of the roentgenograms of the Registry chosen to serve as illustrations are irreprehensible from the standpoint of technique; in many instances the technical points essential for a satisfactory reproduction of the roentgenogram had been neglected.

It is a pleasure to give acknowledgment to all those who in this country and in Canada have helped me in securing the roentgenograms for illustrations. I am greatly obliged to Drs. Codman and Ewing for many valuable suggestions. Thanks are due my chief, Dr. Rowan, without whose support it would have hardly been possible for me to give this study the necessary amount of time and work. I am glad to accord credit to Drs. Beye and Baxter who kindly read parts of the manuscript.

ANATOLE KOLODNY.

CONTENTS

PAGE

INTRODUCTION 1

HISTORY 3

ETIOLOGY 5

CLASSIFICATION 8

OSTEOGENIC SARCOMA:

- Pathology 26
- Clinical Course 67
- Diagnosis 85
- Therapy 107
- Prognosis 119

EWING'S SARCOMA:

- Pathology 126
- Clinical Course 137
- Diagnosis 144
- Therapy and Prognosis 149

MYELOMA 155

UNCLASSIFIED SARCOMA:

- Angio-Endothelioma 163
- Extraperiosteal Sarcoma 169

GIANT CELL TUMOR:

- Pathological Concept and Etiology 172
- Pathology 175
- Clinical Course 189
- Diagnosis 194
- Therapy 202
- Prognosis 210

INTRODUCTION

THROUGH the irony of fate the case which led to the organization of the Registry of Bone Sarcoma proved to be a carcinomatous skeletal metastatic lesion. This error is not surprising when one realizes that according to experienced observers at the time of the organization of the Registry in 1920 in about half of all cases diagnosed by the clinician and pathologist as bone sarcoma the diagnosis was erroneous. At that time the necessity of a collective study of the primary malignant bone tumors, maladies so rarely diagnosed correctly, became clear to Codman. Experience and accumulation of data were needed to study the natural history of this disease and to test the value of the various therapeutic measures. A narrow road was ahead of the Registry. While in most diseases with a grave prognosis the histological information is secured from the postmortem examination, in bone sarcoma we have had, in the majority of cases, an amputation or exploratory specimen on which to base our diagnosis during the life of the patient. This fact together with the characteristic temptation to prognosticate the outcome of the case stimulates an effort to trace back the clinical history for diagnostic signs and symptoms for future guidance. Independent of the diagnosis and prognosis given by the Registry the individual case goes on its natural course and the outcome frequently demonstrates the diagnostic and prognostic error of the Registry leaders. Experience is the main asset of the physician and none is so valuable as that acquired by mistakes and errors in judgment. The work of the Registry based upon a study of a wealth of material unequaled in the history of bone tumors has accomplished an immense destructive and constructive task. The numerous various classifications of artificially segregated types and varieties of bone tumors suggested in the literature have been carefully considered and in the main have been rejected and an attempt was made to offer a basis for a new standard classification. The widespread idea of the sanctity and invulnerability of the pathological diagnosis was undermined and the importance of the clinical and radiological findings stressed. The benignity of the giant cell tumor constantly questioned by the amputating surgeon was definitely proved and the necessity of conservative treatment advocated. The subject of bone sarcoma was freed of many misconceptions in the literature filled with statistical data of hundreds of cases of "bone sarcoma," cured by various therapeutic methods.

It would be erroneous, however, to shut our eyes on all the blanks in our knowledge of this most important subject even though it has been studied for six

years by a most efficacious collective method. Much remains to be learned of the pathology and histogenesis of such important lesions as Ewing's sarcoma or giant cell tumor. In the study of these tumors no use has been made as yet of modern methods of hæmatology, tissue cultures, and vital staining which promise much. Our knowledge of the histogenesis of normal bone marrow leaves much to be desired. Even less have we learned of the purely clinical side of the problem; we must have the courage to confess our failure as yet to master these diseases. The treatment of bone sarcoma is just a part of the most important and live problem in medicine today of combating the malignant tumors. The uncertainty of cure of bone sarcoma by any method, the variety of form, both in relative pathological activity, in clinical location and superimposed changes are sufficient to stir one's interest for purely scientific reasons, not to speak of the outstanding clinical importance of the subject.

HISTORY

THE word "sarcoma" belongs to those time-honored terms which are enjoying free general usage in medicine despite the fact that they do not at the present express the original idea suggested by the sponsors of the term. The name sarcoma has been used to designate a tumor with a firm and fleshy feel. The conception of sarcoma a century ago was very wide including all tumors that could not be grouped with other tumors belonging to the few, separate entities known at that time. Curiously enough with such a wide conception of sarcoma the tumors known today as bone sarcomata were not included in this group. The superficial gross characteristics of these tumors led to the acquisition for them of such names as fungus, exostosis, tophus, osteoma, and bone cancer. Even Virchow who brought light in most of the dark fields of pathological anatomy and who was the first definitely to separate these sarcomata in a special chapter of oncology did not discriminate sharply enough between sarcoma of bone and cancer. It was Boyer who in 1845 christened these tumors with a name which was destined to follow the development of the knowledge of them up until this day. When at about that time a histological trend of study was directed toward malignant tumors, Virchow described the morphology of sarcoma of bone with clarity and detail surprising for that time. A few years later Robin made an attempt to describe two types of bone marrow cells from which bone tumors originate: the myeloplacque, identified by later investigators as the osteoclast, and the medullocell, later osteoblast. Notwithstanding the confusion by Robin of bone forming and blood forming cellular elements, his was an important step forward in the study of bone tumors; and in the renowned monograph of Nélaton of 1860 the author stressed the importance of the discrimination between the *tumeur à myéloplaques* which is clinically benign and the malignant *tumeur à médullocelles*. These correspond to what we know today as giant cell tumor and osteogenic sarcoma. When a few years later Billroth made the erroneous statement denying the clinical and prognostic importance of the histological peculiarities of sarcoma, Gross (1879) severely criticized him in a brilliant monograph on *Sarcoma of the Long Bones* in which he also emphatically stressed the usual benign course of the sarcomata with a giant cell structure. Years have passed during which the knowledge of malignant bone tumors was at a standstill. Then came the illustrious work of Ewing tending to coordinate the clinical and laboratory sides of the question. In America, Ewing, among pathologists, and Bloodgood, among clinicians, have brought it about that the

giant cell sarcomata of bone have been segregated in a separate sharply defined clinical entity as a benign condition. To do away entirely with the term sarcoma in this disease, the latter was named giant cell tumor. About six years ago on the initiative of the ingenious Codman there came into existence the famous Registry of Bone Sarcoma of the American College of Surgeons which has greatly fostered the study of malignant bone tumors. Today the knowledge of bone sarcoma is yet somewhat nebulous and far from being accurate. The treatment of patients with these diseases is still a matter of ardent discussion and study. On the most important chapter on the origin of these tumors today there is only a dim light. Much is left to be learned in this truly virgin field of science.

ETIOLOGY

IT is a surprising failure of oncology that the knowledge of the etiology of tumors is far behind our knowledge of their structure and natural life. Numerous new theories have been evolved during the last hundred years, many of them purely scholastic inventions without any actual facts to support them. Is there any wonder when old theories which have exterminated themselves because of lack of confirmation are brought to life over and over again under different names? Among the factors underlying this failure are the attempts of pathologists to create theories which would explain the origin of all tumors, whether benign or malignant, carcinomatous or sarcomatous. A study of the clinical and pathological nature of a neoplasm suggests that the etiology of each neoplastic entity is probably specific. It is apparent that Ribbert's theory of the displacement of tissue complexes which explains well enough certain tumors, fails in explaining those numerous instances of tumor growth which follow upon irritation. Opening his famous address on genesis of tumors, Billroth said: "Ueber die letzten Ursachen der Dinge nicht nachzudenken, ist im gewissen Sinne ein beneidenswertes Glueck." No theory explains more than the other; none completely satisfies the inquisitive mind. Whatever theory is favored we cannot free ourselves of the acceptance of a constitutional disposition for tumors. In the course of time the hypothesis of a general disposition of the organism to blastomatosis has been generally conceded.

The etiology of the group of bone tumors known as bone sarcoma has frequently been linked with trauma. Whatever the personal views of the investigator as to the etiology of tumors in general may be, it cannot be denied that trauma seems to be frequently associated with the origin of bone sarcoma. Because of the wide difference in definition of trauma by various pathologists the literature is discrepant in statistics about the clinical incidence and the etiological importance of trauma. The leading question suggesting itself is: "What is the relation between the trauma and the appearance of the tumor?" Refraining from a discussion of the many hypotheses of purely philosophical nature, which lack actual basis, I will outline here the belief to which one is inclined after a study of the osteogenic sarcoma group of the Registry material.

In studying a large series of osteogenic sarcoma one cannot fail to be impressed with the fact that all instances of this tumor may be arranged in series in such a way that at one end will be a mass resembling callus, as we see it after fracture, and at the other a most cellular, highly malignant looking osteo-

genic sarcoma. Cases of osteogenic sarcoma may be identified when the bone absorption, new bone formation, and the vascularity can hardly be told from callus in various stages of bone repair. This observation leads one to believe that the development of osteogenic sarcoma is guided by the same principal laws of growth that are usually observed in the animal organism when in the stage of natural growth or when repair and regeneration are on hand. The main and important specific condition required for the development of an osteogenic sarcoma is a stimulus to growth.

From the fertilized ovum the growth capacity is transferred to the animal tissue cells. As long as the organism is in a stage of physiological development, the cells grow without additional stimuli from the outside. But when the growing organism has reached its adult age—the terminal stage of physical development—the growth tendency of the tissue cells is rapidly interfered with, so that no advanced growth is possible. It is a result of what is known as "growth restraint." The enormous rôle of growth restraint is readily understood when one thinks what would become of the animal organism if growth were not interfered with. The tissue cells in adult organisms do not lose their growth abilities, the latter having merely changed from actual kinetic to potential. On this potential growth ability of tissue cells is based the whole process of tissue regeneration and repair. No life would be possible if the potential growth ability of tissue cells to multiply when repair is needed were lost. Trauma with the consequent necessity of repair leads to a temporary change of the potential growth ability to kinetic, i.e., it leads to a temporary elimination of growth restraint in the traumatized region. In some instances, in the presence of rare predisposing factors which are not understood, trauma may lead to a complete loss of growth restraint in the injured organ. The physiologically accelerated regenerative processes may lead then to a violent multiplication of mesoblastic cellular elements which have entirely lost their main object—physiological repair. The continuous multiplication of as yet undifferentiated cells may be followed by dissemination throughout the organism and by the whole characteristic picture of metastasis of a malignant growth. Regeneration and repair are the main but not only effects of trauma, which have some relation to the etiology of tumors. Separation and isolation of cell complexes, hæmorrhage with conseqeunt absorption and encapsulation, necrosis of tissue—they all play an important part in the etiology of mesoblastic tumors, when brought together with predisposing factors.

Thus it is believed that sarcoma of bone is frequently a result of a loss of growth restraint following an accidental or occupational trauma to a previously normal bone or a surgical trauma to an existing benign bone tumor growth. Not infrequently when the tumor appeared immediately after the trauma or when the primary tumor was noticed after a trauma but not long before a generaliza-

tion of the disease was seen, it is probable that the tumor existed long before the trauma and the latter merely led to the discovery of it or an exacerbation of its growth. The mooted question, not how often sarcoma has a trauma history but how frequently a trauma is followed by sarcoma adds to the discredit of the importance of trauma as an adjuvant etiological factor in an organism predisposed to blastomatosis.

CLASSIFICATION

A SATISFACTORY classification of a disease is the main step to an acquisition of knowledge of it. No science is possible if no uniform language and terminology exist. The lack of progress in the knowledge of bone sarcoma in the last decades is not surprising when one realizes the vast number of various names used for bone sarcoma in different countries, in different centers of the same country, and even by different persons in the same institution. The establishment and propagation of a classification of primary sarcomatous tumors of bone is not merely a responsible but also a most difficult task. As in any other branch of science, there are in the knowledge of these tumors certain time-honored prejudices which have been and still continue to be spread among the workers in this field. A name or an idea, however wrong it may be, is difficult to eradicate if it has decades of history behind it. Furthermore a classification will be of practical value only if it is really accessible to the average clinician or laboratory man. This is especially true in bone sarcoma, a condition with a relatively rare clinical incidence.

Two temptations are presented to anyone who is working on a classification of bone tumors. On one hand there is always a desire to be as complete as possible in the classification, so that no bone tumor would be left in the same group with tumors of a slightly different variety, anatomical or clinical. On the other hand one is tempted to sacrifice preciseness for the sake of simplicity. There have been many victims of this last trend of thought. Billroth was one of them when he said: "The subdivisions made according to the histological peculiarities of the various sarcomata, are of no great value during life." It is a matter of fact that no ideal classification of bone tumors is possible with our present-day limited knowledge of their origin and natural history. Every bone tumor is specific in some way or other, and a classification based upon a distinction of the individual fine points would defeat its purpose by the abundance of groups, types, and varieties, which would vary also with the opinion of each expert.

It is not my intention to offer here a new classification. The classification of the Registry of Bone Sarcoma is the best among the prevailing ones. A study of the Registry material, however, suggests certain changes in this classification. The apparently bizarre fact that the Registry material suggests a classification differing from the one accepted by the Registry Committee can be explained by several factors. In the first place the classification of the Registry was accepted

by the Registry Committee when only a limited number of cases were as yet registered and available for study. In the second place the Registry classification was suggested by a Committee composed largely of laboratory men, hence the presence in it of anatomical points of interest without clinical sequence. Further the purpose of the Registry being the study of all bone lesions diagnosed by anyone as bone sarcoma, the Registry classification includes all pathological conditions of bone and not merely primary malignant bone tumors. In the following, the desirable changes in the Registry classification will be brought out by way of a review and critical analysis of it.

The classification accepted by the Registry Committee embraces all bone lesions under eight headings.

1. Metastatic tumors primary in tissues other than bone.
2. Periosteal fibrosarcoma.
3. Osteogenic tumors, (*a*) benign and (*b*) malignant.
4. Inflammatory conditions.
5. Benign giant cell tumors.
6. Angiomata, (*a*) benign and (*b*) malignant.
7. Ewing's tumor.
8. Myeloma.

Since in the present writing only primary sarcomatous bone tumors are dealt with, the following headings alone will be discussed: Heading 2 (periosteal fibrosarcoma), heading 3 subdivision *b* (malignant osteogenic tumors), heading 6 (angioma), heading 7 (Ewing's tumor), and heading 8 (myeloma). The conditions under heading 1, not being primary lesions, and those under heading 4 and the subdivision *a* of heading 3 being essentially benign conditions, will be touched upon merely insofar as it will be necessary to emphasize the clinical, roentgenological, and pathological points characterizing sarcomatous structures as contrasted with these conditions. The benign giant cell tumors will be dealt with separately. To make the matter clearer, the present analysis begins with malignant osteogenic tumors.

The old term osteosarcoma still prevails in the continental literature, where all sarcomatous tumors of bone are embraced under this name. Osteosarcoma, as used in Europe, does not leave out myeloma, Ewing's tumor, and even giant cell tumor, which are all clearly separate clinical and pathological entities. Like "bone sarcoma," osteosarcoma is a collective name which should not be used in the medical literature when scientifically more precise terms are possible today.

Ewing is responsible for the term "osteogenic sarcoma." All primary sarcomatous tumors of bone which do not belong in any of the other groups of the Registry classification are to be referred to osteogenic sarcomata. "Osteogenic sarcoma" includes mainly the lesions which have been known for long as "perios-

teal bone sarcoma," a term which has spread in this country since the days of Gross, and which does not stand a critical analysis as will be brought out later. In free translation osteogenic means derived from bone. The true meaning, however, of osteogenic sarcoma as understood by those who accept the Registry nomenclature is a sarcomatous tumor derived from ancestors of cells which, when duly differentiated, are known as osteoblasts. An osteogenic sarcoma thus is an osteoblastoma, somewhat as a carcinoma of the skin is an epithelioma. Of course, there is a difference in this analogy between osteoblastoma and epithelioma. While in the latter the epithelial cells composing it are more or less typical and duly differentiated, in an osteogenic sarcoma various stages of development of osteoblasts can be seen, from a simple spindle cell to mucoid, cartilage, and even true bone cells.

Simple and clear as this conception of osteogenic sarcoma is, it is of the utmost importance. It involves a series of questions of primary significance for the etiology and pathology of these tumors. Recently in the French literature opposition was raised against this conception of "osteosarcoma" as the authors call these tumors. Departing from a discussion of questions of physiological ossification and the subsidiary rôle of the osteoblasts in it the authors arrive at a modern conception of osteosarcoma. According to their views these tumors are not osteoblastomata, but sarcomata of the connective tissue cellular elements present in the bone and bone marrow. Thus, no difference is to be encountered between a sarcoma of bone and a sarcoma of a fascia. Modern as this conception is it is contrary to well known facts. The authors have seemingly neglected the fact that pulmonary metastases of osteogenic sarcoma show tumor cells, cartilage, myxomatous tissue, and even bone. Since it is impossible to presume that all the numerous metastatic foci have originated from embryological inclusions of which each contained cells of every variety (myxomatous, cartilaginous, and bone), this fact proves that the tumor cells brought through the vascular system from the primary focus into the lungs have retained the ability of the osteoblasts to differentiate in mucoid, cartilage, or bone cells.

A mistake made very frequently is to understand under osteogenic sarcoma a tumor producing bone. An osteogenic sarcoma does not necessarily mean a tumor in which the tumor cells are forming bone. Production of bone is merely a potential ability of the tumor cells. If the tumor is slow growing and the differentiation of the tumor cells is well expressed they may reach the ultimate goal and become bone cells, otherwise the differentiation may desert them halfway, and they are left as mucoid or cartilaginous cells. They may even remain permanently in their primary stage of spindle cells without any definite arrangement and visible aim. While the tumor cells are undifferentiated and rapidly multiplying they affect a corrosive action upon the involved bone. The bone melts away under the approach of the tumor cell. Later on differentiation of

Fig. 1. Case 312.[1] Osteogenic sarcoma. Photomicrograph of a section taken from the same tumor as in Figures 2 and 3. An area showing the structure of a fibrosarcoma.

the tumor cells leads to bone production, but at the same time the undifferentiated tumor areas continue to destroy bone. It is analogous to physiological bone repair in fractures. The first stage of healing of a fracture is the resorption of the bony spicules and the jagged edges of the fragments. Formation of osteoid tissue with calcification is the next stage, when the provisional callus is formed. Then follows the third stage of resorption and consolidation of the exuberant callus. In non-union of fractures, because of local reasons or constitutional disorders the cellular elements which have been called to activity by the law of repair do not differentiate in bone. They remain at the stage of cartilage or myxomatous tissue or even a fibrous septum is left in the line of fracture.

The subdivision of osteogenic sarcoma in various groups and varieties is an exceedingly difficult task. It is obvious that the single criterion of whether a certain peculiarity of a group of tumors is to be sufficient reason for an exclusion of the group in a separate variety or type, is whether or not this peculiarity influences the clinical course of the tumors. Tumors with a histology which

[1] Most of the illustrations are made from material selected from the Registry of Bone Sarcoma of the American College of Surgeons, and the case numbers all refer to the cases recorded in that Registry. Illustrations taken from other sources are so indicated.

Fig. 2. Case 312. Osteogenic sarcoma. Compare with Figures 1 and 3. An area showing atypical cartilage.

seems to influence their prognosis are apparently entitled to be carried as a special variety or type, separately from the main bulk of tumors. The influence of the structure of the tumor upon the mortality and prognosis and therapy is the only indication for a practical and sound subdivision.

There has long been felt a tendency among students to differentiate sarcomatous tumors according to the variety of the tumor cells and the preponderating tissue which the tumor imitated. Naming tumors on the basis of the features of their cells is inaccurate, because such names do not convey any data of diagnostic importance. Round celled, spindle celled, giant celled sarcomata—they all originate from mesoblastic elements and unless information is furnished as to their origin and clinical peculiarities, these terms are mere sounds lacking any diagnostic and prognostic enlightenment. Naming osteogenic sarcoma according to the specific tissues which the tumor is imitating is also of questionable value. The majority of osteogenic sarcomata studied show a conglomeration of various tissues of mesoblastic origin. If one is not satisfied in the examination of an osteogenic sarcoma by a few sections of one area and one makes an exhaustive study of the whole tumor, one rarely if ever fails to encounter all or several of these tissues (Figs. 1, 2, 3). In one tumor fibrous tissue will predominate, in

Fig. 3. Case 312. Osteogenic sarcoma. Compare with Figures 1 and 2. An area showing low grade bone formation in a cellular tumor.

another osteoid, in still another myxomatous, and in others chondromatous or osseous tissue will preponderate. A diagnosis including a combination of all or several of these tissues preceding the words "osteogenic sarcoma," merely adds to the mystery with which bone tumors are surrounded. They fall short as far as enlightenment on the clinical and pathological diagnosis and prognosis is concerned.

From the tissue complexes which are found in osteogenic sarcoma, myxomatous tissue has enjoyed the greatest independence in the past. Ribbert in Europe and Bloodgood in America have consistently intimated that myxoma belongs to a separate anatomical entity with a specific origin. Ribbert insists that myxoma is not a product of degeneration of a higher grade tissue since similar degenerations are unknown in normal life and therefore cannot be expected in tumors. However, there is on hand sufficient evidence against the independence of myxomatous parts of osteogenic sarcomata. The myxomatous tissue there is merely a phase of differentiation of the mesoblastic elements which are the progenitors of osteogenic sarcoma. A typical fibroma on one hand and chondroma on the other are almost always encountered along with the occurrence of myxoma.

Plate 1. Case 571. See Figure 6. Osteogenic sarcoma in a woman 25 years old. Illustrating the inadvisability of differentiation of periosteal, subperiosteal, and medullary osteogenic sarcomata. The tumor involves the entire transverse diameter of the bone and also surrounds the bone extraperiosteally.

The alleged poorer prognosis of myxoma as contended by Bloodgood is probably due to technical difficulties of eradication of these tumors and not due to their higher potential malignancy. A chemical analysis of myxoma demonstrates the relationship of these tumors to chondromata. Since both myxomata and chondromata yield mucilaginous protein which on hydrolysis yields sulphuric acid and glucosamine (chondroitin-sulphuric acid) they should be related.

The Registry classification recognizes four anatomical types of osteogenic sarcoma: (1) The periosteal, (2) the medullary and subperiosteal, (3) the sclerosing, and (4) the telangiectatic. The spokesman of the Registry, Codman, doubts the advisability of this differentiation, but Ewing, after whose ideas the Registry classification is framed, urges this subdivision because these types are supposed to differ in their gross anatomy, in their microscopic picture, and in their clinical course. From an analysis of Ewing's views as far as the periosteal and the subperiosteal and medullary types are concerned a different conclusion is arrived at. These types are evidently grossly anatomical and the main feature which separates these two groups, as one can judge from the names, is the position in the bone, whether periosteal or subperiosteal. It is obvious that this distinction is valueless as far as clinical diagnosis goes. To state that the exact location of a tumor is periosteal as opposed to subperiosteal and medullary is clinically impossible. To depend for such purposes on the roentgenogram is

Fig. 4. Case 214. Osteogenic sarcoma in a man 37 years old. Amputation was done 1 year after the onset. The cortex is grossly well preserved and one is inclined to group it with subperiosteal tumors without a medullary involvement. However, tissue taken from the medullary cavity shows sarcomatous involvement.

unsafe because even if the average fair roentgenogram shows only an involvement of the periosteum or a subperiosteal tumor with an intact periosteum this is not conclusive. Frequently, when an unusually clear roentgenogram is made of the amputated extremity a more diffuse and extensive involvement can be made out than is seen in the routine examination. There is no satisfactory proof that such circumscribed osteogenic sarcomata really occur with an involved periosteum and intact subperiosteal tissue. Furthermore even if such conditions occur theoretically by the time they attract the attention of the patient they are more or less diffuse. Not only is it impossible to state the exact location of the tumor from the clinical and roentgenological examination, but, very frequently even with the gross specimen in hand it is hardly possible to say with certainty where the tumor invasion stops. Thus it is evident that no clear gross anatomical features distinguish the "periosteal" type from the "subperiosteal and medullary." Neither is the microscopic picture of the periosteal type entirely different from the subperiosteal and medullary. This contention is especially true when one excludes from the group of medullary sarcoma all tumors other than osteogenic. The frequent occurrence of various intercellular substances in the same tumor indicates that the disease is essentially one and the same, whether the bulk involvement is in the periosteum or extends more subperiosteally (Plate 1).

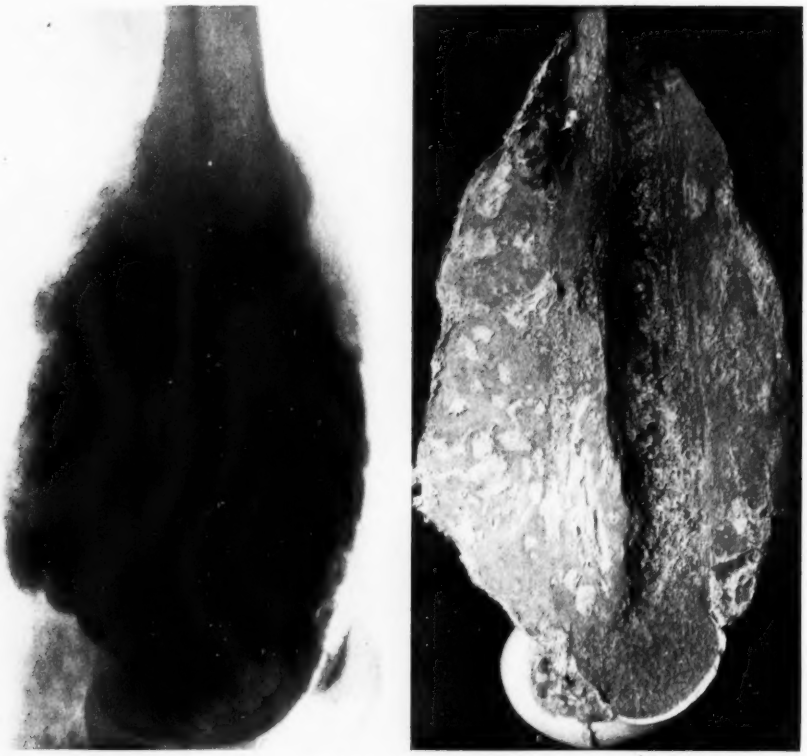


Plate 2. Case 116. See Figure 5. Osteogenic sarcoma in a woman 28 years old. The most outstanding sclerosing tumor in the roentgenogram in the Registry collection. The photograph of the tumor, however, indicates a soft vascular area in the proximal portion of the tumor, and in the medullary cavity. Death with pulmonary metastases 12 years after the onset. Amputation 6 years after the onset and reamputation for local recurrence 3 years later. The roentgenogram was taken 6 years after the onset.

Finally, it goes without saying that a most careful study of the material of the Registry did not furnish any evidence that the prognosis is different in a supposed periosteal osteogenic sarcoma from that of subperiosteal-medullary. In the Registry material if a satisfactory operative and pathological description is given or a good photograph of the gross specimen is present both periosteal and central involvement are seen (Fig. 4). Codman emphasizes this point.

Of all the four anatomical types of osteogenic sarcoma recognized by the Registry classification the "periosteal" has always enjoyed the greatest popularity. Some of the reasons of it have been pointed out above. Another reason is perhaps the fact that an involvement of the periosteum in the roentgenogram is one of the most widely recognized signs of osteogenic sarcoma. To the

Fig. 5. Case 116. Osteogenic sarcoma. The same tumor as in Plate 5. Sclerosing type of osteogenic sarcoma. In proximity to a sclerosing portion of the tumor a cellular area is seen.

inexperienced surgeon or roentgenologist few roentgenograms will suggest the diagnosis osteogenic sarcoma, unless there is the known fan-like structure or at least a lipping of the periosteum—the wedge-like lifting of the periosteum from the cortex of the involved bone. Lipping of the periosteum is a reactive response of the periosteum to bone involvement, and similar lipping is also met with in other than neoplastic bone lesions. However, good roentgenograms from various angles nearly always show it in osteogenic sarcoma and this means that subperiosteal involvement is present.

Investigators who have most consistently urged the recognition of periosteal sarcoma associated this type of bone involvement with the radiating fan-like structure of the tumor in the roentgenogram. It may be said without exaggeration that nothing in the teaching of malignant bone tumors is more popular than this fan-like picture. The idea that this picture is absolute proof of osteogenic sarcoma is widely spread and frequently a clinician will not make the diagnosis of osteogenic sarcoma unless this picture is seen. Facts speak to the contrary; a similar arrangement of the needle-like osteophytes perpendicular to the bone is sometimes found in low grade chronic bone infection, pyogenic or tuberculous.

Plate 3. Case 334. Osteogenic sarcoma in a man 24 years old. The roentgenogram suggests a sclerosing tumor while the gross specimen and the specimen with the injected blood vessels show the exceptional vascularity of the tumor. The roentgenogram was taken 6 months after the onset and one month before a hip amputation was done. Death 10 months after the onset.

Fig. 6. Case 571. Osteogenic sarcoma. The same tumor as in Plate 1. Sclerosing type; close to the acellular tumor mass a cellular area is seen.

Beautiful specimens of macerated bones showing this condition may be seen in the Warren Museum in Boston and in the Army Medical Museum. The presence of these spicules means subperiosteal involvement, as will be seen later, but this is invariably accompanied also by a medullary involvement.

"Sclerosing" is the third anatomical type of osteogenic sarcoma in the Registry classification. The term sclerosing, given by Virchow, means that the tumor sclerosed, the intercellular substance ossified. In the Registry material and also in many cases of private collections studied I saw no case of osteogenic sarcoma in which the microscopic picture of "sclerosis" prevailed throughout the whole tumor (Plate 2). Usually after one or two areas of real sclerosed tumor where only an occasional tumor cell is found, areas are seen where the cellularity of the tumor and the pleomorphism of the tumor cells seem to indicate that no deviation from the grave prognosis of the average osteogenic sarcoma can be expected (Figs. 5, 6). Cases of osteogenic sarcoma studied roentgenologically during the whole natural life of the tumor frequently show at some stage or other, usually in the early beginning a picture simulating sclerosing osteogenic sarcoma (Plate 3). After weeks or months the character of the tumor changes to osteo-

Fig. 7. Case 508. The same tumor as in Plate 18. An exceedingly vascular "telangiectatic" osteogenic sarcoma riddled with large blood spaces.

lytic. Were this tumor examined microscopically in its first osteoblastic stage one would be inclined to group it with the sclerosing tumors, while at a later stage the tumor would turn out to be definitely osteolytic.

The fourth anatomical entity of the Registry classification is the telangiectatic osteogenic sarcoma. As the meaning of the word suggests, this type is characterized by abundant blood vessels and spaces. In the extreme variety of this type when large blood vessels make up the main bulk of the tumor, and when only a thin wall of tumor tissue surrounds these blood spaces, a pulsation of the tumor may be felt (Fig. 7). In the old literature this variety of tumor was known as "bone aneurysm." The walls of the dilated blood channels being very thin and consisting of a single layer of endothelium, it is readily understood why a definitely poorer prognosis is to be expected in such cases; there is an immense opportunity for the malignant tumor cells to break into and to spread through the vascular system to the lungs. Like sclerosis in osteogenic sarcoma telangiectasis is a relative feature, all transitional stages being encountered between tumors with a poor blood supply and exceedingly vascular tumors. Telangiectatic is therefore also relative and not a definite separate type of osteogenic

sarcoma. It is obvious that telangiectatic is a postoperative or postmortem diagnosis; clinically and radiologically this diagnosis is usually impossible. Just as sclerosing is an extreme of differentiation of the tumor intercellular substance, telangiectatic is an extreme in the richness of the vascular supply of an osteogenic sarcoma.

Under heading 2 of the Registry classification is listed "periosteal fibrosarcoma." Under this term a sarcoma is understood which, though intimately related to the periosteum, leaves the cortex of the bone intact. Only very rarely the cortex of the bone is slightly eroded, the erosion taking place at a later date in the progress of the tumor. Thus, these tumors are extracortical, frequently surrounding the bone for a considerable distance. Virchow called attention to the fact that usually these tumors are found at points of bone insertion of tendons and dense fasciæ. The majority of these tumors present a typical dense fibrosarcoma. In the Registry material under "periosteal fibrosarcoma" are grouped also instances in which the origin of the tumor could not be established with certainty. A well ascertained true example of a sarcoma originating from the outer layer of the periosteum is up to the present day unknown in the Registry material. The "periosteal fibrosarcoma" cases registered however bear out the fact that this tumor is of a decidedly better prognosis than osteogenic sarcoma. It is easy to see that the term "periosteal fibrosarcoma," accepted by the Registry Committee, is unsatisfactory for important reasons. The word "periosteal" readily raises in one's mind associations with "periosteal osteogenic sarcoma," a tumor of an entirely different origin, microscopic structure, and mainly different clinical course. The Registry Committee seems to have realized it and has attempted to avoid confusion by adding the word "fibrosarcoma" to "periosteal." V. Rindfleisch has pointed out that the origin of "periosteal fibrosarcoma" is from the "fascicular" layer of the periosteum, while the "periosteal osteogenic sarcoma" originates from the cambial layer. In the literature two other terms are mentioned for this tumor: "parosteal" and "extraperiosteal." Of these two I prefer the latter. "Parosteal" conveys rather an idea of a process spreading along lines parallel to the bone; parosteal sarcoma is a sarcoma of a fascia or muscle rather than of the periosteum itself, while "extraperiosteal" seems to emphasize the origin of the tumor from the "external" fascicular layer of the periosteum.

The sixth division of the Registry classification is "angioma." Two subheadings are given there: the benign angioma and the malignant. Of the second subdivision, the malignant, which alone is to be considered here, no cases are registered with the Committee. The Registrar, Codman, believes that this type, angiosarcoma as he calls it, is exceedingly rare if it exists at all; most angiosarcomata from the literature he regards as telangiectatic osteogenic sarcomata. It is true that much confusion was aroused in our knowledge of tumors

of the vascular system by the term "angiosarcoma." This term, given by Kolaczek, originally was intended for the so-called peritheliomata, tumors originating from the vessel wall and not from the endothelial lining of the vessel. Later this term found its way to tumors with a clear endothelial origin, which are better called angio-endothelioma. Unfortunately the very lax requirements instituted by Golgi and Kolaczek for a diagnosis of an endothelioma have led to frequent diagnoses of endothelioma in cases in which no such condition was present. This fact greatly discredited the angio-endotheliomata as an actual anatomical entity in the eyes of most leading pathologists. The majority of pathologists are unwilling today to diagnose an angio-endothelioma even when all evidence is before them. Because of this skepticism instances of true angio-endothelioma slip by under various other diagnoses. This is also true of angio-endothelioma of bone. During the study of the Registry material I found two cases of primary angio-endothelioma of bone which had been passed by the majority of examiners as metastatic tumors. Angio-endothelioma of bone is a malignant tumor of the vascular system of bone with a typical microscopic structure and a definite origin from the endothelial lining of the vessel wall.

I realize that the rarity of skeletal angio-endothelioma would not justify a segregation of this condition as a separate clinical entity in a classification of practical clinical importance. In a large series of malignant bone tumors a careful study will occasionally mark instances which will be difficult to classify according to their anatomical structure and clinical behavior. Obviously such instances cannot be all brought out as clinical entities if the classification of such an obscure condition as bone sarcoma is to remain clear and digestible. Such instances must be pigeonholed awaiting accumulation of more accurate knowledge of bone tumors in general. To such a group of "unclassified tumors" it would be well to carry the angio-endothelioma of bone which although a clear anatomical entity is too rare to justify a separate heading in the classification. It is probably best to list under this group also the extraperiosteal sarcoma (periosteal fibrosarcoma of the Registry classification), true representatives of which seem to be very rare. To this group should belong also such questionable cases as No. 420 and No. 349 of the Registry collection, the first a tumor resembling somewhat a giant cell tumor in the primary and pulmonary metastases but not typical enough of a true giant cell tumor structure, and the latter a true giant cell tumor which, following multiple operations and ill advised therapeutic procedures, was transformed into a distinct malignant tumor and gave pulmonary metastases.

The seventh division of the Registry classification is "Ewing's tumor." The knowledge of this entity, scarce as it is, is unfortunately limited only to those who have studied the Registry material. This fact however is not surprising when one recalls that only in 1920 was this tumor established by Ewing as an anatomical and clinical entity among the malignant bone tumors. Pathologists

unfamiliar with bone tumors have been for long submerging and still continue to submerge Ewing's tumor under the diagnosis of round cell sarcoma or solitary myeloma. A specific clinical course, a typical gross anatomy, and a unique histology serve as the basis for the existence of this entity. In studying the Registry material even without any thought of classifying the tumors it is surprising with what ease the impression grows in one's mind that these tumors represent a clear-cut separate disease. This tumor affects young patients, rarely above 21 years of age. It frequently begins with a clinical picture resembling acute osteomyelitis, a reason why in the majority of cases this mistake in diagnosis is usually made. The response of the tumor to radiation, whether roentgen-ray or radium is remarkable; however, permanent cure from radiation is as yet unknown. Metastases to other bones is a characteristic feature of the terminal stage of this tumor in divergence with osteogenic sarcoma in which bone metastases are very infrequent. The histological structure of the tumor is at variance with any variety of osteogenic sarcoma. Ewing suggests the origin of the tumor from the endothelium of the bone marrow. In connection with this he suggests a wider scope of cellular elements for the term "endothelium," since he admits that the vascular endothelium bears no relation to the origin of the tumor. Deferring for the present time a detailed discussion of this question it will suffice to say that no proof has as yet been accumulated to support Ewing's hypothesis of the endothelial origin of this tumor. The term "endothelioma" under which this tumor frequently appears tends to confuse it in the minds of the occasional observer with angio-endothelioma. If Ewing's tumor is in any way related to the endothelium of the bone marrow it is an "endothelial myeloma" and it should not be called "endothelioma." It is most satisfying to see that the Registry classification has refrained from either term, endothelioma or endothelial myeloma. However the term Ewing's tumor decided upon by the Registry Committee is not well chosen and is incompatible with the intention of the Registry to reserve the word "tumor" for a giant cell sarcoma which is essentially benign. In giant cell sarcoma the word sarcoma has been replaced by the word tumor in order to emphasize the benign nature of the lesion. The lesion understood by the Registry Committee under Ewing's tumor is a true malignant condition and the retaining of the word sarcoma would cause less confusion as to the clinical behavior of the tumor. For this reason I am of the opinion that Ewing's tumor should be substituted by Ewing's sarcoma until a time when we learn more of the nature of this condition, more of its origin and its morphological ancestry. For the present, whatever the morphological nature and the origin of this tumor, it is undoubtedly a clinical entity and as such it is entitled to an independent place in the classification.

"Myeloma" occupies the eighth division in the Registry classification. This is a tumor originating in the medullary cavity of a bone, appearing simultaneously

as numerous nodules ranging in size between the size of a pin head and that of a walnut. The large majority of myelomata are multiple tumors, only one solitary tumor having been recorded by the Registry during the six years of its existence. This tumor is held to be derived from the specific bone marrow cells of the myelocyte series. Myeloma is the only true round cell tumor occurring in bone. Because of its multiple character myeloma was recognized long ago as a systemic disease, and this recognition led to a view that myeloma is not a true tumor but is to be regarded as in some relation to the pseudoleukæmic or aleukæmic group. For purposes of classification there is no need to enter a discussion of all the fine details of the derivation or etiology of myeloma. There are characteristic features which stamp myeloma as different from osteogenic sarcoma. Myeloma is a bone marrow tumor characterized by multiple foci of origin, by frequent albumosuria and rare metastases, frequently affecting short bones, and especially favoring the ribs, sternum, vertebræ, and skull. When situated in long pipe bones, it favors midportions rather than the ends of the bones. Myeloma recedes rapidly under radiation but no permanent recovery by radiation is recorded. In many of these features myeloma resembles Ewing's sarcoma, from which it is easily distinguished because of its morphology and different radiological appearance. The wide destruction of the bone marrow leads to early cachexia and anæmia, and because of the rapid resorption of bone, fractures and infractions occur early and frequently. Attempts at subdivision of myeloma into separate types according to a variation in morphology has met with much opposition. The present day knowledge of the fine points of morphology of myeloma is limited. Subdivisions according to histological features offer no therapeutic indications. Despite the divergence in the histological structure all these types belong essentially to the same disease entity.

As a conclusion to the above discussion all primary malignant bone tumors can be subdivided in the following groups:

1. Osteogenic sarcoma.
2. Ewing's sarcoma.
3. Myeloma.
4. A group of unclassified sarcoma including among others such "near entities" as angio-endothelioma and extraperiosteal sarcoma.

No subdivisions of the main groups in types is recommended. Slight as are these changes of the Registry classification insofar as sacrifice of accurate knowledge for the sake of simplicity is concerned, they are important because of the clearness with which they present the classification of this most confused chapter of oncology. That these changes are justified theoretically was pointed out in the analysis of the Registry classification. From the practical point of view there can be no doubt that these changes render the classification of the primary malignant bone tumors more understandable for the roentgenologist and patholo-

gist and especially for the clinician, who is ordinarily relatively inexperienced in bone tumors.

This modification of the Registry classification is not to be considered as wholly satisfactory and final. No clear cut or hard and fast rules are possible in dealing with such a confused problem as malignant tumors of bone. There is no assumption on my part that in the future with the advance of our knowledge of these tumors it will not be found advisable to subdivide these four entities into groups and types with a peculiar histology, origin, and clinical picture. When a classification of a group of pathological conditions, as in bone tumors, precedes the acquisition of exact knowledge of these conditions the only way a classification can proceed is the deductive one, by dividing the general group into narrower and more specific classes. This explains why sometimes bone tumors are encountered when it is difficult to include them in any of the divisions of the classification. In the present classification these tumors are grouped under the "unclassified." The number of individual cases in this group is not large and with the advance of our knowledge will decrease still more. Occasionally the question will arise whether to group them with one or the other division. These are the so-called borderline cases, which, with all clinical and radiological data and the gross specimen before him, and with the section under the microscope, one hesitates to place in any certain group. The question of the decision of the exact nature of a borderline case is especially difficult when the diagnosis rests between a malignant tumor with a very grave prognosis and a benign condition, whether a tumor growth or an inflammatory process. In these cases the pathologist's answer is most decisive and therefore most responsible as far as the therapeutic and especially prognostic indications are concerned. *Natura non facit saltum* and there may be expected all transitional stages from a benign condition to the most fatal tumor growth. No classification can with advantage consider similar problems as long as our knowledge of bone tumors is so meager, and our experience so fallacious. However, at present I agree with Codman that any pathologist who has studied the Registry material with care will make a satisfactory diagnosis and prognosis in over 90 per cent of the cases presented to him, provided the data are technically good.

OSTEOGENIC SARCOMA

PATHOLOGY—*Gross Anatomy*

THE pathology of primary malignant tumors of bone embraces the gross anatomical picture of the tumors and their histological structure. Unlike other pathological conditions the gross anatomy of these tumors is more complicated because of their location in such a dense unyielding organ as bone. Because of the great resistance to the spread of the tumor offered by the bone and also because expansion of the bone alone is far insufficient for a progressive growth of the tumor, the gross picture of these tumors is exceedingly peculiar. This is especially true of osteogenic sarcoma—that main group of primary malignant tumors of bone.

As was pointed out in the chapter on classification a distinction between central, subperiosteal, and periosteal osteogenic sarcoma is unfounded. An osteogenic sarcoma is a diffuse process of the involved bone and there is no way to determine precisely the origin of the tumor, i.e., whether medullary or periosteal. Of course there is always a period in the history of an osteogenic sarcoma when the most active growth of the tumor is especially pronounced in one certain area of the involved bone, whether it be the medulla, cortex, or periosteum. It is evident that the gross picture will be influenced as to whether the tumor grows more rapidly in the region of the medullary cavity or close to the periosteum. In the first case the bone cortex will exercise resistance to the spreading of the tumor for a much longer period of time than in the last case, when the periosteum will be reached and perforated by the tumor at a much earlier date. It is evident therefore how erroneous it is to accept as the origin of the tumor the center of the tumor mass, which in many cases even late in the disease is confined by the cortex although the latter may be perforated in many places.

To understand the capricious features characteristic of osteogenic sarcoma one has to keep in mind the main factors which influence the gross picture, shape, and consistency of these tumors. The foremost place among these factors is occupied, on one hand, by the continuous aggression of the tumor elements and on the other by the defensive protective measures of the involved bone against the growing tumor. The degree of differentiation of the tumor elements, the vascularity of the tumor and the regressive changes in it are factors greatly influencing the consistency of the tumor.

The aggression of the tumor cells and the defense of the involved bone may be noticed in every case of osteogenic sarcoma. The natural instinct of the tumor—to grow and to increase in size—leads to more or less destruction of the bone in the involved area. Destruction of the bone to a greater or lesser degree is a condition *sine qua* there is no osteogenic sarcoma. The attempts of the tumor to extend are directed in two ways—one is toward the outer aspect of the bone and the other is along the medullary cavity. This latter direction is a point of less resistance, because of the loose structure of the bone-marrow and also because the endosteum does not exercise as much activity to protect the medullary cavity from invasion of the tumor as the periosteum does against perforation of the cortex into the surrounding tissues. This explains the fact that cases of osteogenic sarcoma are seen in which the medullary cavity is literally stuffed with tumor from end to end while the radiograph indicates a tumor wholly limited to the area of involvement of the cortex and periosteal reaction.

The self defense of the involved bone is taken over by the periosteum. The periosteal reaction in osteogenic sarcoma is the only morphologically evidenced attempt of the organism to protect itself against this extremely malignant condition. The periosteal reaction is especially well expressed when the tumor is located in its most frequent situation—the metaphysis of a long pipe bone. It is much less striking when the tumor is located in the shaft or in those rare cases when it is situated in the epiphysis proper. The periosteal reaction of a bone involved with an osteogenic sarcoma consists in formation of new bone, apposed on the outer surface of the cortex of the involved bone. This apposition of new bone leads to a gradual thickening of the bone. Gradually the tumor breaks its way through the old cortex by way of the Haversian system and encroaches upon the newly apposed periosteal bone which it gradually displaces. Slowly the tumor spreads subperiosteally along the surface of the bone. The periosteum is being pushed away from the underlying bone by the newly produced protective osseous tissue and especially by the growing tumor mass. Nothing interferes with this elevation of the periosteum except the defensive lipping to be described later until the stripping off of the periosteum reaches the epiphyseal line. Because of the most intimate attachment of the periosteum to the epiphyseal cartilage here the elevation of the periosteum comes to a stop and further growth of the tumor leads to a club shaped enlargement of the epiphyseal end of the spindle. It is of sufficient diagnostic importance to remember that in osteogenic sarcoma the old cortex is frequently seen passing through the tumor mass appearing grossly intact. Permeation rather than perforation of the cortex expresses better the pathological process. In this, osteogenic differs from Ewing's sarcoma, in which the bone generally is broken up and displaced by the tumor.

The gross structure of the tumor mass consists of stalactite-like rods of hyaline osteoid and osseous tissue arranged like the spikes of a wheel perpendicular to

the long axis of the involved bone or like fluffy trabeculæ spread beneath the periosteum on top of the cortex of the involved portion of the bone. In the radiograph the first structure is represented by the classical sun-ray-like appearance.

Not infrequently the periosteal reaction is expressed in only slight bone production. This is common in osteogenic sarcoma of the shaft. The rapidly enlarging tumor leads to an elevation of the periosteum from the involved bone in the manner outlined above, but the periosteum is unsuccessful in forming a true bony capsule about the tumor. Only at the polar ends of the grossly fusiform swelling which are farthest from the most active portion of the tumor, the periosteally new formed bone has temporarily escaped destruction. In the radiograph this new formed bone with a jagged edge gives the picture of "lipping," which is a cardinal sign of osteogenic sarcoma not yet sufficiently appreciated (Plate 4). Physiologically this lipping is a result of stretching of the periosteum by a mass intruding between the bone and its periosteum. In a periosteum put under tension the osteogenic capacity is greatly stimulated. Hence such a lipping is sometimes encountered in merely traumatic conditions when a hæmorrhage strips away the periosteum from the bone. Thus, while lipping is a sign of malignancy, it is not in itself a result of malignant tissue. In the extremely rapidly growing osteogenic sarcoma lipping is less distinct, the lips are poorly formed because of a lack of time for their formation and also because of the extremely intense bone destruction.

In cases with a rapid growth of osteogenic sarcoma and a weak periosteal reaction pathological fractures are encountered. Such fractures at once give an outlet to the expanding tumor by tearing the periosteal capsule. The tumor then enters the ultimate phase in its local development, infiltration of the surrounding soft tissue. More frequently, however, than through a pathological fracture, an osteogenic sarcoma reaches this final stage by way of perforation of the periosteal capsule. The continuous stretching of the periosteum by the tumor expanding beneath it finally leads to a perforation of the periosteum at some point. The tumor changes its main direction of expansion along the bone to a centrifugal infiltration of the surrounding tissue. A longitudinal section through a tumor at this stage shows the involved bone with the tumor mass spreading along it, intramedullary and subperiosteally, and externally to the periosteum, the most recent tumor mass connected with the main tumor by a pedicle through the perforation. In perforation through the periosteum of osteoblastic osteogenic sarcoma, when the subperiosteal portion of the tumor consists of the stalactite strands of hyaline osteoid and osseous tissue, the extraperiosteal portion of the tumor loses this radiation structure and the osteoid and osseous tissue of the tumor here has no evident orderly arrangement.

After perforation of the periosteal capsule has taken place, nothing more stands in the way of the aggressive tumor. Muscles are stretched, thinned, and

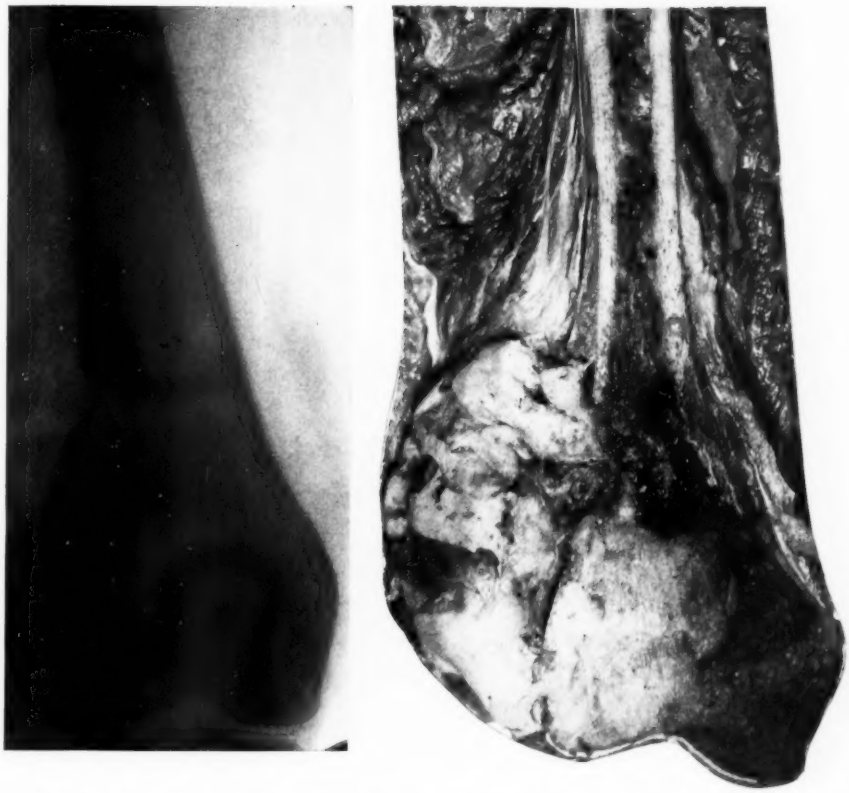


Plate 4. Case 43. Osteogenic sarcoma in a girl 17 years old. Osteolytic variety. The photograph shows invasion of the epiphysis and lipping of the periosteum.

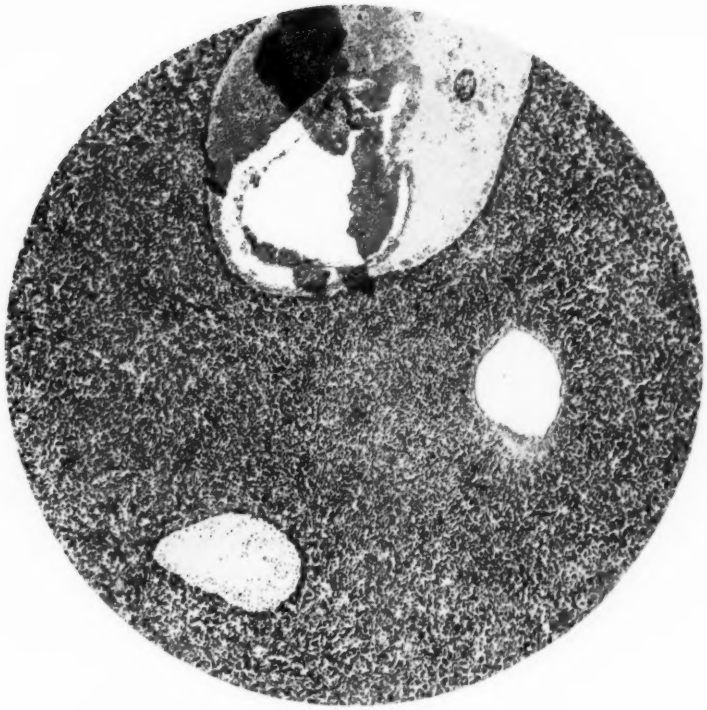
atrophied so that it is not unusual to find the tumor covered merely with glossy skin. Ulceration of the overlying thinned out skin is unknown in osteogenic sarcoma; it is simply stretched and atrophied. Even more resistant to osteogenic sarcoma than skin is cartilage. Articular cartilage and cartilage of the epiphyseal junction offer more resistance to the aggressive tumor than any other tissue in the human body (Fig. 8). The best explanation of it is probably the absence of vascular channels in cartilage. As a matter of fact in adults with ossification of the epiphyseal line an involvement of the epiphysis by an osteogenic sarcoma of the metaphysis takes place relatively early, while in young individuals an extension involvement of the epiphysis is more likely to take place by the indirect route of perforation of the metaphyseal periosteum and secondary involvement of the epiphysis from without than by direct extension through the car-

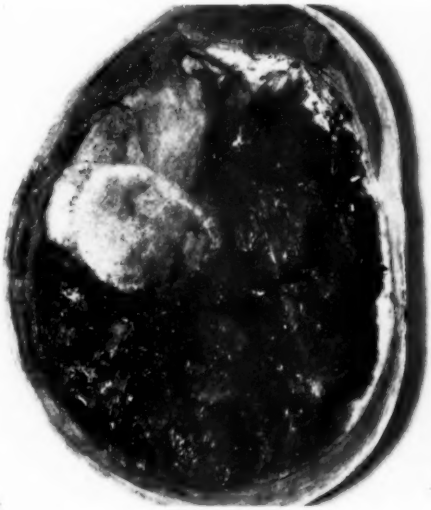
Fig. 8. Case 408. Osteogenic sarcoma. Illustrating the resistance of the articular cartilage to the tumor invasion. A layer of spongiosa is seen between the tumor mass and the articular cartilage.

tilaginous epiphyseal line. In approaching the articular cartilage the tumor destroys all the bone, leaving a thin layer of spongiosa which separates the cartilage from the tumor mass. In this respect osteogenic sarcoma differs from giant cell tumor where the cartilage is frequently in direct contact with the tumor. However instances are known when the entire articular cartilage *en masse* was found separated from the underlying bone destroyed by the tumor which was infiltrating the joint cavity.

Perforation of an osteogenic sarcoma into the joint with an actual involvement of the articular synovia is not rare in far advanced cases complicated with a pathological fracture or by surgery in a region close to the joint. More unusual is a spontaneous involvement of a joint without a preceding pathologic fracture. After perforation into the joint cavity the tumor grows freely and a subsequent involvement of an adjoining normal bone is known to occur.

It is easy to see that an exploratory incision or incomplete local excision of an osteogenic sarcoma readily changes the entire course of the disease. The perforation of the periosteal capsule, long awaited by the tumor, is brought about in these cases by the surgeon himself. Articular involvement in similar cases is not infrequent.





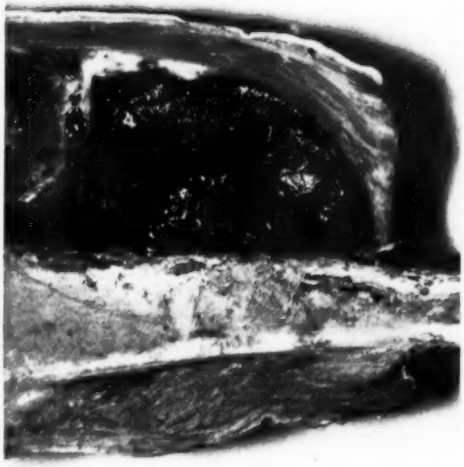


Plate 5. Case 98. See Figure 28. Pulsating osteogenic sarcoma; "malignant bone aneurysm" of the old authors.

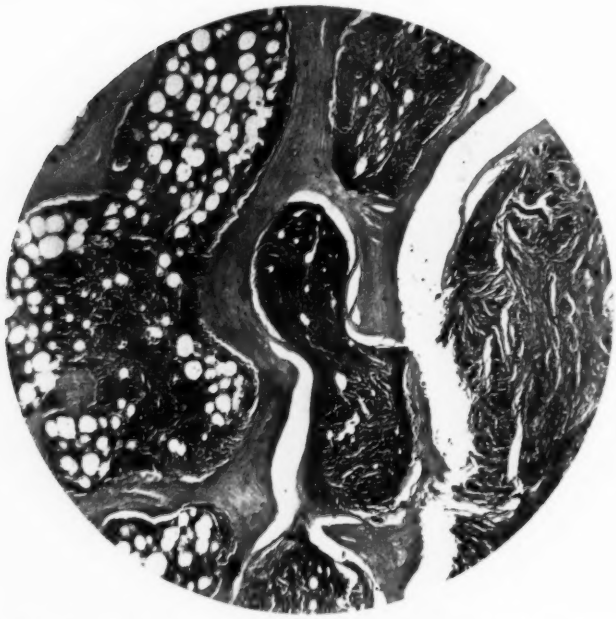


Fig. 9. Case 623. Osteogenic sarcoma. Showing the structure of the so-called medullary fibrosarcoma; an osteolytic variety of osteogenic sarcoma.

The consistency of an individual osteogenic sarcoma is influenced chiefly by the degree of differentiation of the elements constituting the tumor. The great variety in consistency an osteogenic sarcoma may attain is best understood if one thinks of all the changes in consistency which may be presented by immature callus in which the differentiation was interrupted at some stage of development. There are all the transitional stages from the very first attempts of organization of the blood clot between the bone ends through the stage of fibrosis and cartilage formation up to consolidated bony callus. Our knowledge of the morphology of bone repair and regeneration is too meager to enlighten us on the origin and the various stages of the development of an osteoblast. There is, however, considerable evidence tending to show that fibrous tissue, myxomatous tissue, and cartilage are definite stages through which osseous tissue may pass during its development. These definite stages are met in osteogenic sarcoma. In some, the differentiation of the tumor elements goes on to formation of fibroblasts, in others it is sidetracked to myxomatous tissue, in still others the ability of the cells to differentiate goes no further than the stage of cartilage, while in some osseous tissue is formed. The consistency of osteogenic sarcoma, however, is not

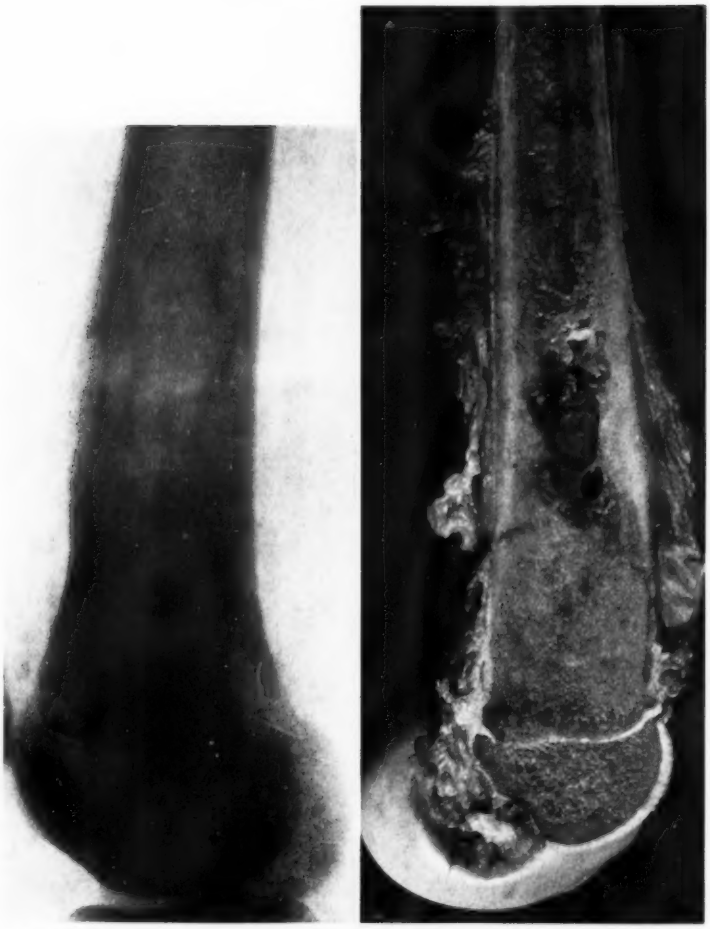


Plate 6. Case 341. Osteogenic sarcoma in a boy 15 years old. Notice the cyst in the gross specimen; a result of central degeneration of the tumor. Death 1 year and 10 months after the onset.

defined by the presence of one certain type of tissue—in the majority of cases usually two or more various tissues may be observed simultaneously.

According to the degree of differentiation of osteogenic sarcoma these tumors are usually divided in two groups: osteoblastic and osteolytic depending upon the degree of bone production or destruction. From the material studied I believe that there are no pure osteoblastic or pure osteolytic osteogenic sarcomata. The tendency of all cases of this class of sarcoma is to produce bone in

Fig. 10. Case 584. Osteogenic sarcoma in a girl 19 years old. Amputation was done after heavy radiation therapy. Notice the extensive cyst; the cyst was filled largely with necrotic tumor tissue.

some part of the tumor, because, as it was pointed out above, the cells of which an osteogenic sarcoma is composed are of the osteogenic variety. An osteolytic tumor, having destroyed much of the involved bone, usually shows minute foci of new formed bone sparsely scattered through the tumor, which when the tumor is cut give a gritty sensation. In an osteoblastic tumor we see along with masses of new formed bone, casting the characteristic dense shadow in the roentgenogram, also numerous minute foci of cellular areas without any trace of bone formation. There is, however, a decided difference in the consistency of an osteoblastic and osteolytic osteogenic sarcoma. The softness of an osteolytic osteogenic sarcoma ranges between the firm elastic consistency of cartilage and the semifluid consistency of myxomatous tissue. Besides minute foci of bone,

Fig. 11. Case 313. Skeletal chondroma. The tumor is incised and the central portion laid open. Showing extensive degeneration with cyst formation in the cartilaginous matrix.

Fig. 12. Case 462. Small spindle cell variety of osteogenic sarcoma.

osteolytic tumors are composed of cartilage, myxomatous tissue or fibrous tissue, or a combination of two or all three. The consistency is dependent upon the prevalence in the tumor of any one of these tissues.

From the standpoint of the theory of "repair and growth restraint" osteolytic osteogenic sarcomata may be regarded as tumors in which the differentiation of the cellular elements has proceeded to a certain point on the long path between the primitive mesoblast and the mature osteoblast. In relation to this two observations are interesting. One is that osteolytic tumors are much more frequent in adults than in children and adolescents; and the other is that an osteolytic osteogenic sarcoma more frequently invades the epiphysis than an osteoblastic osteogenic sarcoma. The latter fact is probably merely a result of the former—the ossified epiphyseal line in adults is not resisting to an epiphyseal invasion of a metaphyseal osteogenic sarcoma. The fact that osteolytic osteogenic sarcomata are met more frequently in adults in whom the regenerative powers of the bone are weaker than in the young seems to correspond well with the expressed idea of an osteolytic tumor being an undifferentiated osteogenic sarcoma.

The consistency of an osteogenic sarcoma with the preponderance of fibrous tissue is not very different from that of a fibroma, especially of the sclerosed

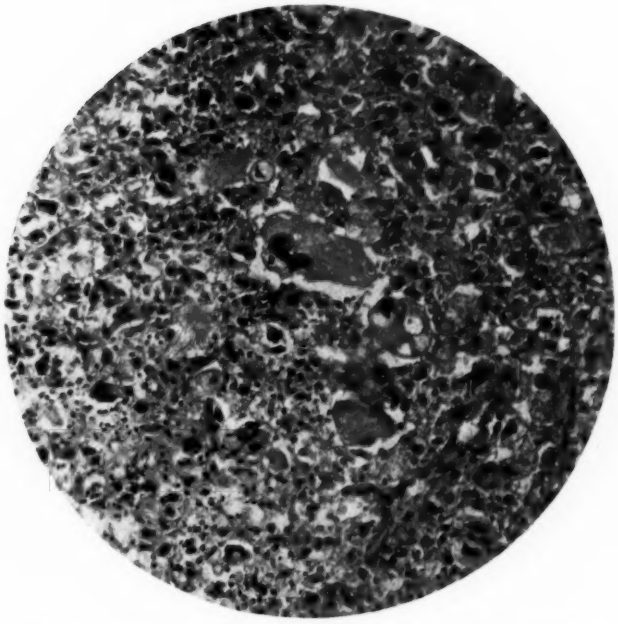


Fig. 13. Case 540. Osteogenic sarcoma. Showing the large polyhedral cells, said to be undifferentiated cartilage cells.

variety. An extensive destruction of bone with a substitution of a large fibroma is extremely rare, only two cases having been recorded in the Registry. Osteolytic tumors with prevalence of fibrous tissue are frequently located close to the medulla and this fact has been brought (Phemister) as evidence of the necessity of the recognition of "medullary fibrosarcoma" as a separate anatomical entity. The constant findings in such tumors of traces of bone formation, however, seem to contradict the rationality of a separation of these tumors from the group of osteogenic sarcoma. The clinical course and ultimate results furnish hardly any evidence of the exclusiveness of this variety of osteogenic sarcoma. Histologically a medullary fibrosarcoma is an exaggerated osteitis fibrosa in which the development of fibrous tissue is so extensive that it leads to an atrophy of the cancellous bone (Fig. 9).

When cartilage prevails in the osteolytic form, the tissue is firm, elastic, gritty, with the grayish white, semitranslucent, milky, bluish appearance of cartilage. Osteogenic sarcomata entirely consisting of myxomatous tissue are uncommon. More frequently myxomatous areas are found in tumors with an abundant admixture of cartilage. The presence of myxomatous tissue in the tumor is frequently accompanied with hæmorrhagic extravasation and this adds

Plate 7. Case 49. Osteogenic sarcoma. Showing the huge amount of chromatin in the tumor cells passing through a mitotic stage.

Fig. 14. Case 62. Osteogenic sarcoma. Showing mononuclear and polynuclear giant cells in a spindle cell stroma.

to the conspicuousness of the gross appearance of the tumor: blood clots, contrasted with soft, jelly-like, translucent, grayish-white tissue.

The fact that cellular areas can be found in definitely osteoblastic osteogenic sarcomata is of interest in relation to the idea, widely spread among bone pathologists, of a sclerosing type of osteogenic sarcoma. If true sclerosing osteogenic sarcomata do exist they are apparently a great rarity. In careful investigation of a tumor with the most pronounced dense shadow typical of sclerosing osteogenic sarcoma in the roentgenogram, cellular areas may be found with growth propensities typical of most malignant tumors. Cases have been observed in which along with a definite sclerosing character of the tumor in the roentgenogram cysts were present. It is best perhaps to draw an analogy between sclerosing osteogenic sarcoma and scirrhous carcinoma: the density of the stroma does not negate the presence of carcinomatous tissue.

The influence of the degree of vascularity of an osteogenic sarcoma on its consistency and gross anatomy is evident. In tumors with prevalence of cartilage and myxomatous tissue the blood supply is very moderate. In such cases the vessels usually run beneath the capsule of the tumor, the central portion of the tumor being poorly supplied. This is especially true of skeletal chondromata

Fig. 15. Case 22. Photomicrograph of section taken from the same tumor as in Figure 16. Showing a lawless conglomeration of tumor cells; the intercellular substance is scant.

which have for many years remained benign and in which an incomplete surgical operation or trauma leads to an increase of the blood supply. Osteogenic sarcomata, which appear like such from the start, are usually very vascular; it is perhaps an analogy to the fracture-hyperæmia which is seen in young immature callus. With the further growth of the tumor the vascularity may change. It may decrease, especially in osteolytic tumors; or it may increase quantitatively and qualitatively—the number and size of the blood vessels may increase.

It has long been urged that cases with excessive vascularity be made a separate clinical and anatomical subdivision of osteogenic sarcoma. The main anatomical peculiarity of such a tumor is its rapid destruction of the involved bone. The destruction may extend along one of two ways: the shaft is simply absorbed and the whole tumor is enveloped in the periosteum with its thin bone capsule, or the shaft is rapidly perforated by the expanding tumor and is broken up in pieces. The remnants of the shaft may be found as sequestra surrounded by blood clots. The amount of tumor tissue present in such an osteogenic sarcoma varies greatly. Usually the cellular areas are quite extensive. In such cases there are no large blood spaces, but numerous small blood capillaries forming a

Fig. 16. Case 22. Compare with Figure 15. Osteogenic sarcoma. Showing an arrangement of the tumor cells in fasciculi; a visible amount of intercellular substance is present.

web-like net throughout the mass of tumor cells. Rarely in an osteogenic sarcoma the tumor tissue present is very limited and the entire tumor is constituted of thin walled alveoli surrounding wide blood spaces. Such tumors rapidly perforate the periosteal capsule, and then they may pulsate and even have a bruit (Plate 5). Despite the view commonly held that the telangiectatic osteogenic sarcomata give a poorer prognosis, there is no sufficient clinical evidence to bear it out. Neither clinically nor radiologically is it possible to make the diagnosis of telangiectatic osteogenic sarcoma. Only after the gross specimen is seen can such a distinction be made. The arguments for distinguishing telangiectatic osteogenic sarcomata as a separate entity are old. In 1879 Gross spoke of telangiectatic osteogenic sarcoma. His definition of this type of tumor was "sarcoma with excessive vascularity." The same definition though changed in words is still being given by all those who are urging the recognition of telangiectatic osteogenic sarcoma at this day. Vascularity alone is not sufficient to justify an exclusion of a tumor which is essentially a typical osteogenic sarcoma. It goes without saying that the definition "excessive" vascularity is very arbitrary and relative.

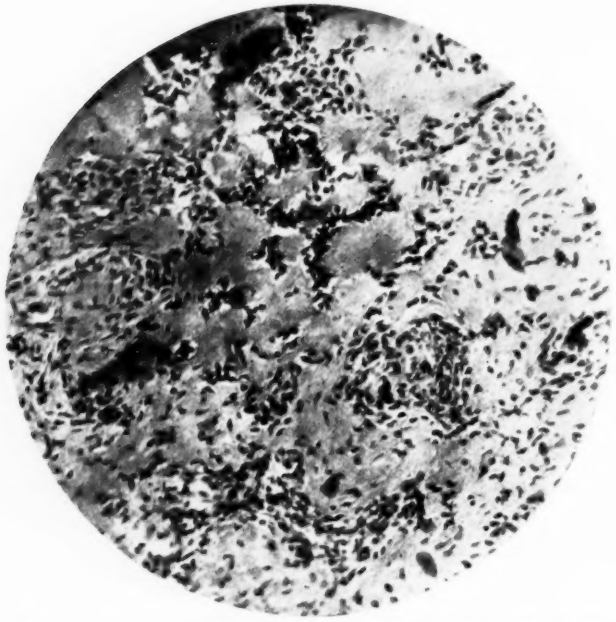


Fig. 17. Case 10. Osteogenic sarcoma. Showing a palisade-like arrangement of the tumor cells about hyaline masses.

The gross anatomical appearance of an osteogenic sarcoma may be greatly changed by regressive changes in the tumor. Because it is of importance to be able to diagnose an osteogenic sarcoma from the gross appearance, knowledge of the frequent degenerative changes occurring in these tumors is of interest. It should be kept in mind that these tumors are prone to degenerate with the result that the color may attain various shades from true fish-flesh to dark-bluish or reddish brown. The consistency may vary from that of the normally encountered resilient feel of liver to that of brain tissue, thin gelatinous substance, or even to fluid. Grossly necrotic tissue, mushy bone-marrow and even a pus-like mass mixed with bone sand are not uncommon findings. It is thought that the cause of degeneration is lack of living material in the rapidly growing tumor, but as a matter of fact degenerative changes frequently occur in very vascular tumors. Central medullary degeneration in osteogenic sarcoma may lead to cyst formation with subsequent perforation of the cortex and periosteal capsule and formation of an extensive extra-osteal tumor (Plate 6). Such tumors have been known for long as "malignant bone cysts," a loose term not in any way expressing the true nature of the tumor. It should be borne in mind by the pathologist and the clinician that radiation therapy also frequently leads to

Fig. 18. Case 537. Osteogenic sarcoma. The tumor cells are surrounded by a hyaline intercellular substance ("near osteoid substance").

extensive degenerative changes and cyst formation (Fig. 10). The regressive changes following radiation of osteogenic sarcoma are so frequent and often so extensive that they deserve more attention from the pathologist and surgeon than is usually given.

Of interest are the gross anatomical features of the so-called recurring type of skeletal chondroma. The clinical features of this not infrequent tumor will be pointed out in detail in their respective places; suffice it to mention here that these tumors are characterized by their long duration before the onset of symptoms. An apparently benign tumor remains stationary for years until an incomplete surgical operation or some other kind of intervention (trauma) leading to an increase of vascularity of the tumor takes place, when the tumor rapidly acquires the characteristics of an osteogenic sarcoma. Grossly these tumors do not seem to involve the cortex at all. However, local recurrence and later metastases and death prove the malignant nature of the tumor. The tumor frequently reaches a very large size. The tumors have apparent capsules which are intimately attached to them. On the cut section, cartilage with calcified areas is seen; this explains the grating with which the knife cuts the tumor. Degenera-

Fig. 19. Case 216. Same tumor as in Plate 11. Osteogenic sarcoma. Showing the polymorphic variety of the cells in atypical "abortive" cartilage.

tive changes with cyst formation are frequent findings in these tumors (Fig. 11). That the pathologist and surgeon are not sufficiently informed about this type of tumor is judged from the instances of erroneous prognoses given.

Structure

The histological picture of the group of tumors designated as osteogenic sarcomata is most conspicuous and varied. It would be erroneous to attempt to give a typical picture of an osteogenic sarcoma. The large majority of osteogenic sarcomata are histologically atypical, or better still there is no one type of osteogenic sarcoma. Osteogenic sarcoma is a tumor derived from cells which are descendants of mesoblastic elements predestined embryologically to form bone, hence the cellular elements of osteogenic sarcoma have the potential ability to differentiate into osteoblasts. The ladder which leads the undifferentiated mesoblast up to the culminating point of differentiation—the osteoblast—has many rungs. The theoretical potential ability of the tumor cells to differentiate expresses itself in reality not in every individual tumor to the same degree. The apex of differentiation—the osteoblast—is the final goal which can be reached

Fig. 20. Case 197. Section taken from the same tumor as in Figure 21. Osteogenic sarcoma. Showing atypical cartilaginous matrix with a beginning myxomatous degeneration.

by a tumor cell but which is not always reached. Something prevents the cell from further climbing up the ladder of differentiation and drives it to assume its lawless career. In almost every case of osteogenic sarcoma cells may be found which are arrested in their differentiation at various steps of the ladder.

It is easy to see how the numerous possible combinations of cells in various stages of differentiation lead to the many varieties of osteogenic sarcoma. The histological picture is still further complicated by the great variety of the intercellular substances. Here also several stages may be encountered in which the intercellular substances vary from simple fibrillation and hyalinization to true bone formation. The changes in the involved bone itself contribute greatly to the complexity of the histological structure of osteogenic sarcoma. The destructive and productive changes in the bone as a result of the aggression of the tumor are most complicated and interesting. Finally, the variety of the blood vessels and other peculiarities of osteogenic sarcomata emphasize the exceptional nature of their histology, as compared with other malignant tumors.

Through the collective studies of "bone sarcoma" by men associated with the Registry, it is possible today to state most definitely that two types of cells

Fig. 21. Case 197. Compare with Figure 20. The myxomatous degeneration of the cartilage is completed.

which have enjoyed a most important place in the histology of osteogenic sarcoma according to former teachings do not really belong to the structure of osteogenic sarcoma. One of these is the giant cell of epulis type. Tumors consisting mainly of this type of cells are permanently excluded from the group of "bone sarcoma." The other type of cell is the round cell. It cannot be emphasized too strongly that osteogenic sarcomata are never of the round cell type. In other words, if the leading cell of the tumor is a round cell it is not an osteogenic sarcoma. Erroneous statements on the presence of round cells in osteogenic sarcoma are frequently due to the fact that the study given the tumor was not careful enough. This is especially true when the predominating cell type is the small spindle cell. In this the nucleus is small and hyperchromatic, and the cell borders are made out with difficulty, so that on cross sections such cells will easily simulate round cells.

The most frequent cell type in osteogenic sarcoma is the spindle cell. This is not surprising when one recalls that the spindle cell is the standard type of cell participating in normal bone repair processes. The most frequently encountered spindle cell is of the small variety with a small hyperchromatic nucleus with undistinguishable cell borders (Fig. 12). When the small spindle cells

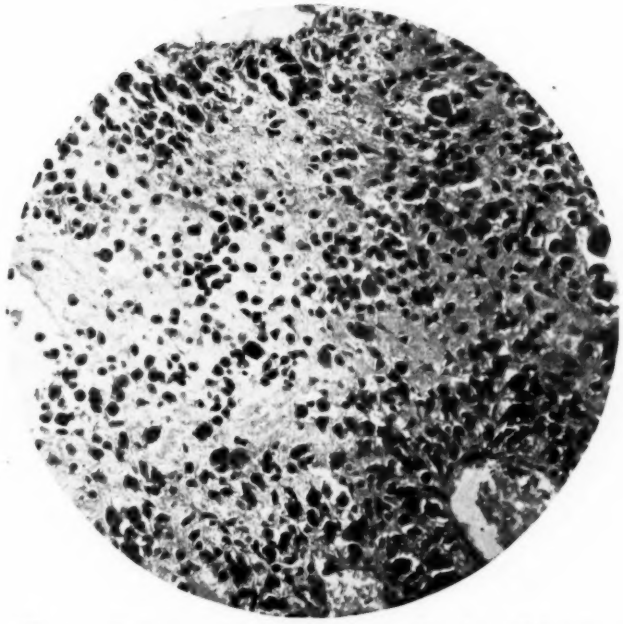


Fig. 22. Case 303. Osteogenic sarcoma. Showing cellular areas about the blood vessels and the relatively acellular cartilaginous matrix.

become larger, thicker, fuller, and plumper with bodies drawn out into filamentous processes we have the large spindle cells. With the further increase in size of the nucleus and the amount of the cytoplasm of these large spindle cells, when the filaments become detached from the cell, we have the polyhedral cells. There is very little morphological difference between such a polyhedral cell and a true bone cell. As in other rapidly growing malignant tumors one of the main characteristic features of the tumor cell is hyperchromatism. The chromatin in the nucleus—that morphological indicator of the growth propensity of the cell—is present here in large amount and causes a deep staining of the nucleus (Plate 7). It should be remembered, however, that hyperchromatism is of significance only when demonstrated in good technical hands; it is then a more important sign of malignancy than differentiation of the cells into fibers and bundles is a sign of benignity. Frequently the hyperchromatism observed is a result of old hæmatoxylin which requires a prolonged decolorization in acid alcohol. Variation in the size of the cells in the same tumor is very frequently encountered in osteogenic sarcoma. The chief feature of an osteogenic sarcoma arising in a persisting or recurring skeletal chondroma is the variation of the cartilage cells. The large polyhedral tumor cells frequent in tumors with a cartilaginous matrix

Fig. 23. Case 559. Osteogenic sarcoma. An early stage of ossification. The individual cells are surrounded by deeply staining granules marking the cell borders.

are said to originate from cartilage cells, or better, are undifferentiated cartilage cells (Fig. 13). Tumors of the great trochanter are noted for such cells.

Of much confusion has been the question of giant cells in bone tumors. Be it said at this time, and this applies to all types of bone tumors malignant and benign, that we have in the distinction between "tumor giant cells" and "giant cells of epulis type" a morphological feature of prime importance. It is not in the scope of this monograph to deal with fundamentals of general pathology with reference to histological distinction between these two kinds of giant cells. While giant cells of the epulis type may be met in most reparative processes of bone and in benign tumors and inflammatory processes, tumor giant cells are seen only in malignant tumors and especially in osteogenic sarcomata. The most frequent tumor giant cells are the so-called mononuclear giant cells, while polynuclear tumor giant cells are seen chiefly in very rapidly growing osteogenic sarcomata (Fig. 14). A warning should be inserted here as to the importance of finding of giant cells in tumors in which an exploratory operation was done previously; the benign granulation tissue in the infected operative wound in the explored tumor may easily show such findings. While not a con-

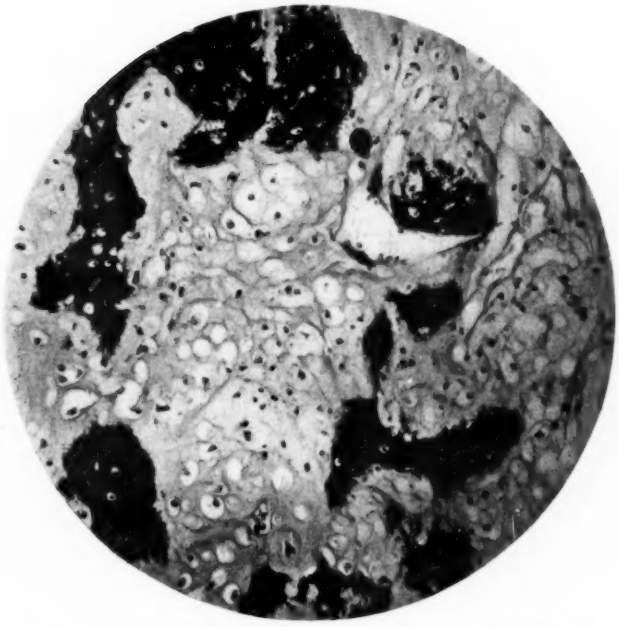


Fig. 24. Case 84. Osteogenic sarcoma. Further stage of ossification. The fusion of the granules leads to formation of bone trabeculæ.

stant finding, giant cells of epulis type are not infrequently seen in osteogenic sarcomata. Their numbers are sometimes so large as to awaken doubt in the inexperienced observer in the diagnosis of osteogenic sarcoma. In such cases a diagnosis of giant cell tumor is usually entertained. Another error is sometimes made when a tumor containing abundant giant cells and arising from tendon sheaths of fingers and toes is regarded as an osteogenic sarcoma of a phalanx. Giant cells of the epulis type are met frequently in far advanced cases of osteogenic sarcoma as a result of rapid and extensive bone absorption. These cells are not infrequent also in necrosing and suppurating areas of osteogenic sarcoma.

The chief morphological difference between osteogenic sarcoma and other malignant tumors is its intercellular substance. It is evident that with our methods of study of the intercellular substance being the same as those employed for study of the cellular elements we cannot expect to be greatly enlightened on this subject. If biochemistry is an integral part of biology, as indeed it is, it is here in the study of the intercellular substance that biochemical methods of investigation are urgently needed. Our knowledge of the intercellular substance, gained solely from morphological studies, is interesting in that it presents more clearly the whole process of development of osteogenic sarcoma.

Fig. 25. Case 273. Osteogenic sarcoma. Showing various stages of neoplastic ossification: fibrillation, hyalinization, and calcification.

All the varieties and shades of the intercellular substance encountered in osteogenic sarcoma may be grouped with one of the five types—hyaline, osteoid, cartilaginous, myxomatous, and osseous. To the hyaline type belongs not solely the hyaline homogeneous mass frequently seen in osteogenic sarcoma. The transparent sclerosed collagen fibers—the product of the filaments of the cell ends on one hand, and the homogeneous amorphous gelatinous mass on the other hand are best grouped with the pure hyaline intercellular substance. That the latter is a product of differentiation of cells normally striving toward bone production is evidenced by some interesting structural features of the tumors, especially well studied in the tumors of the spindle cell variety.

Sometimes in one and the same tumor, and always in a series of tumors, the leading tumor cell can be seen in one of four various arrangements. The first and most frequent finding is when the tumor cells form a lawless conglomeration without any visible order and without any seeming purpose of existence; the intercellular substance then is very scant and may be demonstrated only by special methods of study (Fig. 15). The next stage is when the cells evince some signs of beginning differentiation. The cells become arranged with their long axis in fasciculi, abundantly interwoven and interlaced among themselves

Fig. 26. Case 476. Osteogenic sarcoma. Showing intracartilaginous ossification in tumors with a cartilaginous matrix. Defibrillation of the matrix is followed by ossification.

(Fig. 16). They show a remarkable disposition to retain their arrangement, to which the easily demonstrable collagenous intercellular substance greatly contributes. These are the "bastard" osteoblasts. The third stage is a palisade-like arrangement of the tumor cells about hyaline masses. In cross section such groups of cells present easily distinguishable acini-like structures filled with hyaline substance readily stained with eosin light pink (Fig. 17). Further follow-up of these cells shows the last stage when the cells become surrounded by hyaline substance tending constantly to become more concentrated and by this time to show unmistakable signs of calcification (Fig. 18). The hyaline intercellular substance is a "near osteoid" substance from which the true osteoid matrix may form. In some cases of osteogenic sarcoma the less matured form of "near osteoid" substance prevails—a homogeneous amorphous gelatinous substance which is frequently erroneously interpreted as œdema of the tumor. "Near osteoid" substance is not an exclusive attribute of osteoblastic osteogenic sarcoma; it is not infrequently found in radiologically true osteolytic osteogenic sarcoma, an additional argument against attempts to make osteolytic tumors, mainly the medullary fibrosarcoma an entity distinct from osteogenic sarcoma.

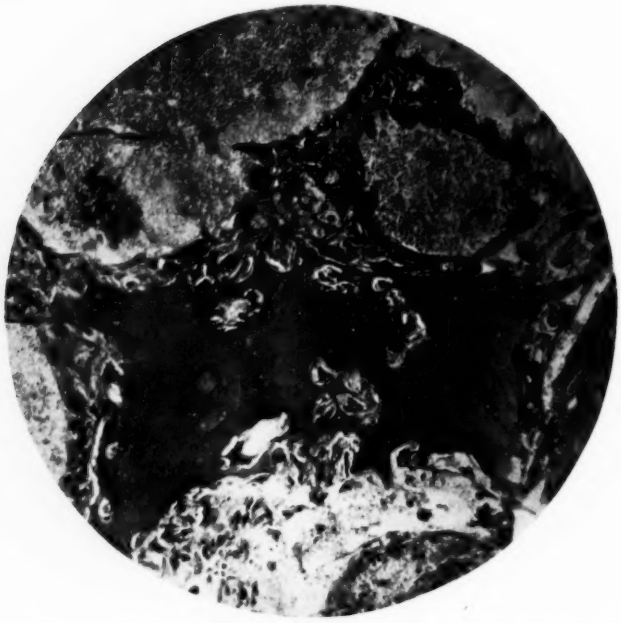


Fig. 27. Case 483. Illustrating the various modes of bone destruction in osteogenic sarcoma: bone destruction due to peripheral absorption, to vascular pressure, and to canalization by tumor cells.

When the hyaline matrix is abundant from the onset, it usually contains cartilage cells of varied size and shape; the ovoid and fusiform cartilage cell spaces contain one or more cartilage cells, mostly without capsule (Fig. 19). This is an abortive kind of cartilage when the pleomorphic character of the cells is the main sign of malignancy. Myxomatous tissue in osteogenic sarcoma is usually seen merely as a phase in tumors with an atypical cartilaginous matrix preponderating (Figs. 20 and 21). An intermingling of various types of intercellular substance in the same tumor is not uncommon in osteogenic sarcoma. The fact that all the tumor cells in the same tumor do not reach the same degree of differentiation at a given time easily explains why the same tumor produces condensation in the roentgenogram in one area and rarification of the shadow in another. The product of the cells of the same tumor may be osteoblastic, chondroblastic, and fibroblastic.

In osteogenic sarcoma with a preponderating relatively acellular cartilaginous matrix cellular areas may be seen closely surrounding vascular channels and spaces. These channels, usually blood capillaries, are in the center of the cellular areas while farther to the periphery avascular abortive cartilage is seen (Fig. 22).

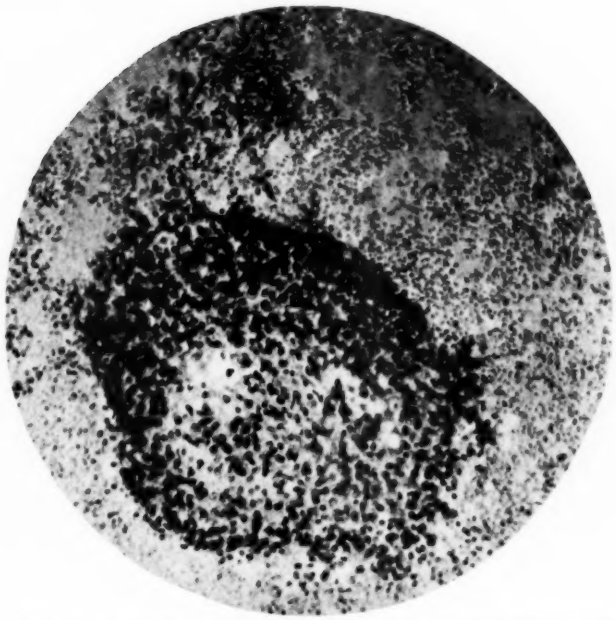


Fig. 28. Case 98. Same case as in Plate 5. "Telangiectatic" osteogenic sarcoma. Showing an island of tumor tissue loose in a blood space.

Whether these are conglomerations of tumor cells merely as a result of the proximity of a nourishment supply (vascular space), or whether the vessel is secondary to the accumulation of cells in one area, is a matter of speculation. In this connection it is interesting to note that the most frequent regressive changes in cartilage cells are the hydropic changes which are probably due to a discharge of water by the cell with precipitation following in it of water soluble constituents.

Some tumors which previously were believed to originate from periosteum, evince a remarkable disposition to ossification of their intercellular substance. They are sometimes known under the confusing name of osteoid sarcoma. These firm or soft medullary, brain-like, semifluid, or gelatinous tumors are traversed by numerous dense glistening striæ which are closely aggregated at their base on the bone surface. These spicules are mostly arranged perpendicularly to the surface of the involved bone; occasionally they pursue a course parallel to the periosteum raised from the bone by the expanding tumor. The fact that this parallel striation, although infrequent, does occur in osteogenic sarcoma, was entirely neglected by previous investigators.

The best explanation of the radiating striation in some osteogenic sarcomata is furnished by topographic-microscopical studies of such tumors. Microscopi-

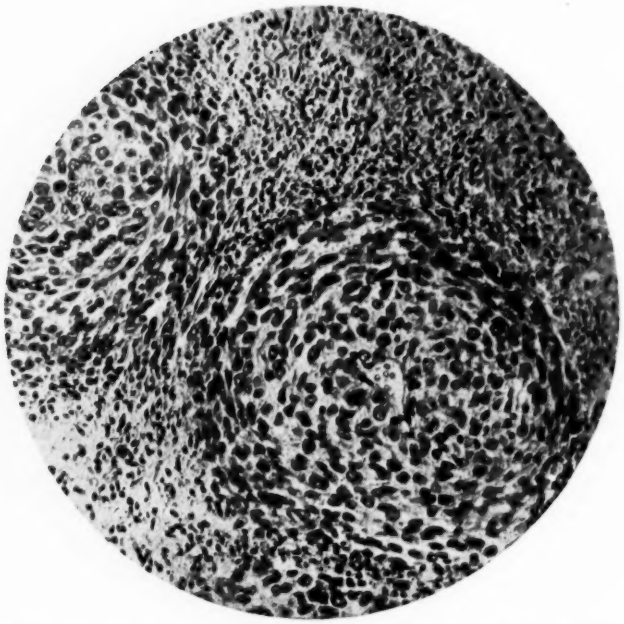


Fig. 29. Case 393. Osteogenic sarcoma. Showing the perithelial arrangement of tumor cells. The cells are in intimate proximity to the endothelium of the thin walled capillaries.

cally one sees dainty spicules attached by a relatively broad base to the cortex of the bone running perpendicularly to the elevated periosteum with its thin bone shell forming the investing capsule of the tumor. After approaching the periosteal bone shell, the spicule changes its course to one parallel with the periosteal bone trabeculæ, finally merging with the latter. The perpendicular striæ are connected among themselves by means of jagged, short, lateral ramifications. The striæ and their ramifications consist of osteoid or true osseous tissue. Tumor masses with thin walled blood vessels running parallel to the perpendicular striæ fill up the spaces between the spicules. From serial sections one gets the impression that the perpendicular blood capillaries emerge from the haversian system of the involved bone. When the periosteum is slowly being pushed away from the underlying bone by the expanding tumor the periosteal blood vessels entering into and arising from the haversian system are stretched in the direction perpendicular to the bone. Apparently the course of the blood capillaries defines the lines along which the osteoid tissue formation and ossification is to take place. That the blood vessels do play an important rôle in the arrangement of the striæ is suggested also by the fact that when the tumor perforates the investing perios-

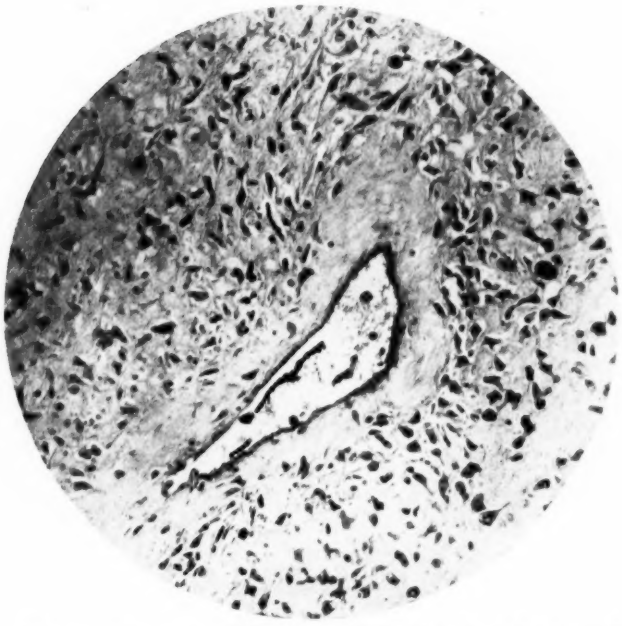


Fig. 30. Case 317. Osteogenic sarcoma. Showing a blood vessel lined by endothelium with a wall consisting of a homogeneous mass suggesting an osteoid or near osteoid substance.

teal capsule and becomes extra-osteal the radiating arrangement is mostly missing in the extra-osteal portion of the tumor with its disorderly arranged blood vessels.

Ossification of the intercellular substance in osteogenic sarcoma is the apex of differentiation which may be reached by the tumor cells and intercellular substance. The whole biological process of ossification is immensely complex and our knowledge of it at present is far from complete or satisfactory. Like normal ossification in the animal body, ossification in osteogenic sarcoma is known to us mainly in its morphological features which certainly are less important than the biological and microchemical features of this process. It is the degree of ossification in osteogenic sarcoma that draws the line between osteoblastic and osteolytic forms. After all, the whole difference is probably a chemical one; that it is not anatomical one sees from the rapid ossification which frequently sets in in an osteolytic osteogenic sarcoma after radiation.

According to morphological findings one meets in osteogenic sarcoma with two types of ossification—the metaplastic and neoplastic. When under certain favorable conditions, of the nature of which nothing is known today, an organic component added to the pre-existing fibrillar intercellular substance leads to

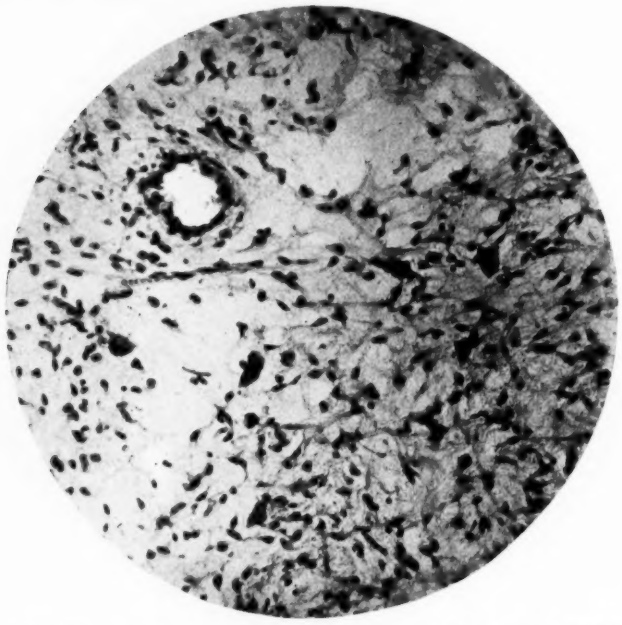


Fig. 31. Case 360. Osteogenic sarcoma. A blood vessel with a wall consisting of a myxomatous substance is seen.

homogenization and hyalinization of it with further calcification taking place, we have osseous tissue formed in the metaplastic way. The successive stages of the development of metaplastic osseous tissue is best studied on small islands of osseous tissue where the process of this form of ossification is in its very early stages. In sections through such areas in tissue undergoing metaplastic ossification one sees about the spindle cells embedded in a homogeneous intercellular substance numerous small cocci- and bacilli-like granules staining with hæmatoxylin deeply blue and with Van Gieson red. The granules are more numerous along the cell borders and decrease in numbers further to the periphery, disappearing entirely at some distance from the cell borders. One sees a picture resembling numerous small magnetic fields of influence with the cells in the centers (Fig. 23). At a later stage the granules continue to multiply and to join each other and soon the whole field is covered by a mass deeply blue in color with pale bone lacunæ occupied by the bone cells (Fig. 24). A certain constant relationship between this granulation and the blood capillaries can be made out. The blood vessels form the centers wherefrom the granules spread continuously. However, the immediate surroundings of the blood vessels are always free of granules even in the final stage of confluence of the granules and bone formation.

Fig. 32. Case 100. Showing a focal lymphocytic infiltration of an osteogenic sarcoma. (A 5 years' cure after amputation.)

That these granules are not precipitated calcium may be concluded from the fact that they are present in the sections after complete decalcification of the tissue. Seemingly the granules belong to the organic substances which are to be added to the pre-existing fibrillar intercellular substance for the process of metaplastic bone formation. The dependence of the granules upon the cells which they surround is seen from the fact that their number decreases with the increase of the distance from the cell. This would suggest that the granules are a product of a biological function of the cell; a function which appears in the cell as a result of some other biochemical process entirely unknown today.

The neoplastic ossification differs from the metaplastic chiefly in that in the former the fibrillar base for ossification is not pre-existing but is formed in the process of ossification by way of cell proliferation. Focal proliferation of spindle tumor cells, arrangement in rows with fibrillation and hyalinization and finally calcification are the main stages of neoplastic ossification in osteogenic sarcoma (Fig. 25). To the neoplastic ossification belongs also the not infrequent intracartilaginous ossification in osteogenic sarcoma with a rich cartilaginous matrix. The cartilage cells arrange themselves in columns separated by bundles of homogeneous fibrils invading the cartilage from the adjoining osteoid tissue

Fig. 33. Case 378. Showing hyalinization of the stroma of a lymph node metastatically involved in osteogenic sarcoma.

(Fig. 26). This defibrillation of the cartilaginous matrix precedes final ossification. In sections one frequently sees small cartilage islands surrounded by recently ossified tissue. In the course of time the remnants undergo the same fate while the older ossified portions slowly change their deeply blue color to a rose pink.

It would be erroneous to believe that it is always possible to draw a strict line between metaplastic and neoplastic ossification in general and especially in osteogenic sarcoma. It is not surprising to find in osteogenic sarcoma, that most perplexing malignant tumor of the human organism, not merely combinations between metaplastic and neoplastic ossification but also instances in which one can hardly decide where the neoplastic process stops and the metaplastic begins. After all, both these terms neoplastic and metaplastic are based merely upon morphological and not biochemical data.

Great importance has been ascribed to the discrimination in osteogenic sarcoma between bone formed by the tumor elements and that formed by the involved bone proper. In the literature and in the discussions on the Registry classification one finds the first referred to as tumor bone as contrasted with the latter as normal bone or reactive bone, i.e., bone formed as a result of a reactive

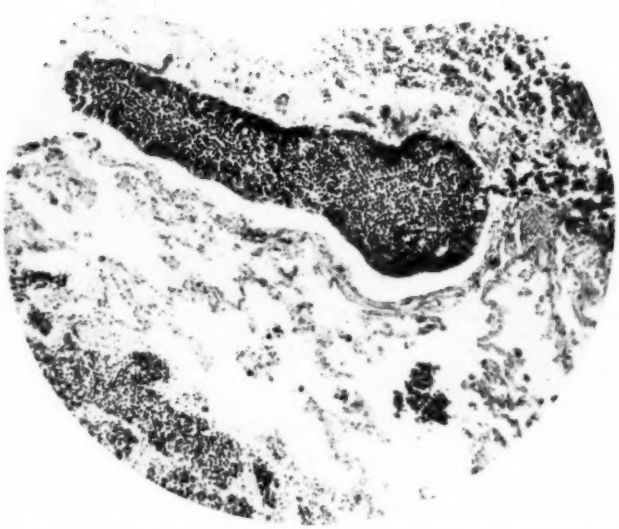


Fig. 34. Personal observation; case not registered. Pulmonary metastases in osteogenic sarcoma. Showing small nodules of tumor beneath the visceral pleura.

inflammatory process of the involved bone due to the presence of the tumor. There can be no two opinions about the fact that reactive bone as such does exist; it is a result of a proliferative reactive inflammatory process in the bone as a response to the involvement; on the other hand the whole discussion about tumor bone and reactive bone does not seem to be backed by morphological or biological evidence. The crux of the argument concerns the presence of two kinds of new formed bone in an osteogenic sarcoma—lamellary bone and trabeculated. Under lamellary bone one understands bone composed of parallel lamellæ supplied with typical bone cells inclosed in bone lacunæ. This is the characteristic structure of the physiologically normal bone. The trabeculated type of new formed bone shows irregular, mostly homogeneous or fibrillar osseous trabeculæ, staining deeply blue with hæmatoxylin, usually lacking the lamellary structure. For long the general belief was that tumor bone is always trabeculated as contrasted with reactive bone which is always lamellary. This discrimination against trabeculated bone apparently is founded on the erroneous statement of Kaufmann many years ago, which identified lamellary bone with bone produced by osteoblasts (*osteoblasten Knochen*). That reactive new bone may be

Fig. 35. Case 457. Osteogenic sarcoma. Advanced pulmonary metastases.

trabeculated we see in new bone formation in metastatic carcinoma, all the new formed bone being reactive bone and not tumor bone. Trabeculated bone is frequently seen also at some stage or other of callus formation in the process of normal fracture healing. On the other hand I have seen cases of osteogenic sarcoma in which areas of bone formation in the pulmonary metastases could well serve as illustration of normal lamellary structure of bone. Apparently the age of bone has much to do with the lamellation; in the slowly developing bone in pulmonary metastases there is ample time for the lamellary structure to take place, while in the very active process of callus formation much bone remains for a time trabeculated. It is not to be understood that in my opinion tumor

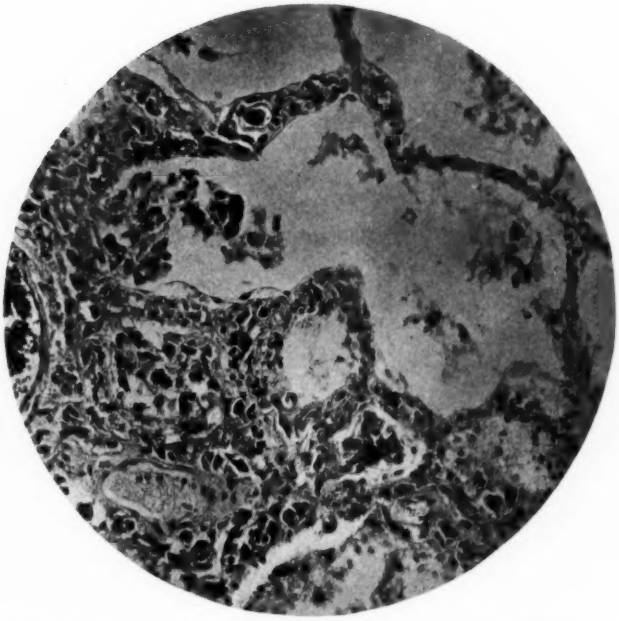


Fig. 36. Case 610. Same case as in Figures 37 and 38. Osteogenic sarcoma. Showing pulmonary interalveolar capillaries stuffed with tumor cells.

bone and reactive bone are entirely identical in every way; it is my belief that with the present day meager knowledge of the ossification process and the inadequacy of purely morphological studies of ready formed bone there is no dependable way to distinguish between tumor bone and reactive bone.

In general there is no essential demonstrable difference in the morphology between the normal physiological apposition of bone and the reactive or defensive production of bone. It was thought erroneously that the new bone is formed from the old. This conclusion is based upon the fact that wherever formation of new bone is following closely absorption of trabeculæ of the old bone, the new bone is seen closely attached to the old surrounding it. A distinction between the old and new formed bone is best reached after staining with hæmatoxylin—the old is red and the new is blue. In cases with very active new bone formation one can frequently find large groups of numerous osteoblasts—"osteoblast reserves" still unarranged but ready to defend the bone against the tumor. These polyhedral cells with relatively large nuclei appear suspicious to the inexperienced, and diagnoses of malignancy in giant cell tumors after radiation have been based upon the presence of such groups of cells along the regenerating bony capsule.

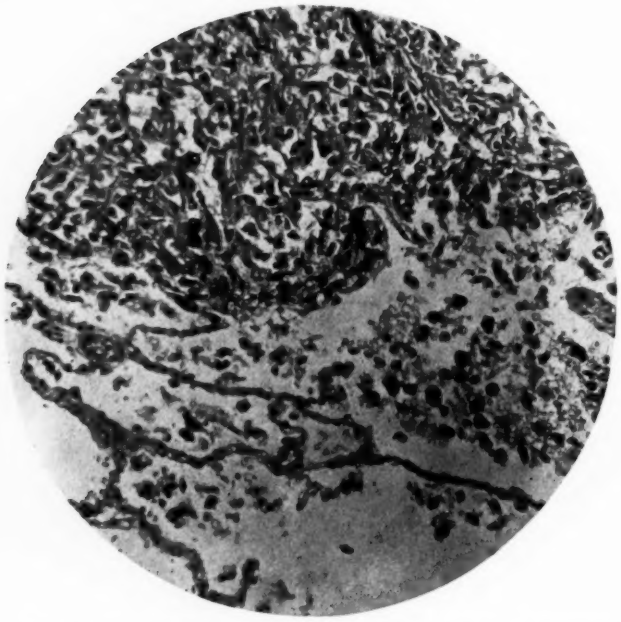


Fig. 37. Case 610. Compare with Figures 36 and 38. Pulmonary metastases with alveoli filled with tumor cells; the alveolar texture of the lung is obscured.

The inelasticity of the normal bone would prevent the growth and expansion of an osteogenic sarcoma if it were not for the destructive process exerted by the tumor upon the involved bone. The absorption of bone under the aggression of an osteogenic sarcoma is accomplished in several ways. One way is the lacunary absorption of the bone; this is the way occurring in the normal process of bone resorption. In osteogenic sarcoma, however, the lacunæ are more flat and small than in normal absorption. This difference is due to the fact that instead of large osteoclasts the tumor cells are found in the lacunæ. That the tumor cells are capable of absorption of normal bone is not at all peculiar to sarcomatous cells; in certain inflammatory conditions one frequently encounters absorption of bone without the typical osteoclasts. Typical polynuclear osteoclasts are usually seen when the destruction of bone proceeds slowly. Absorption may proceed simultaneously from the periphery and from the center of bone trabeculæ. The osteoclasts in the central area are tumor cells which have reached their place by way of newly formed canals as pointed out later. Another means of bone destruction in osteogenic sarcoma is canalization of the bone by advancing blood vessels. These are the Volkmann's canals existing in normal bone but

Fig. 38. Case 610. Compare with Figures 36 and 37. Pulmonary metastases with bone formation.

more numerous in a bone involved by an osteogenic sarcoma. Destruction of bone is also a result of an extreme vascularization of the tumor mass. Direct pressure of the numerous pulsating blood channels on their bony capsule leads to a rapid destruction of the bone. This explains the rapid destruction and not infrequent sequestration of large portions of the involved shaft in very vascular, so-called telangiectatic osteogenic sarcoma. Similar to bone destruction by canalization with blood vessels is that by canalization with columns of tumor cells. Through such new formed canals as well as through numerous new and old blood vessels and through the normal haversian system tumor cells cause minute communications between the medullary cavity and the subperiosteal space, leaving an apparently grossly intact shaft. By all these means the tumor gains ground and space in the involved bone (Fig. 27). The bone cells, released from their seclusion in the bone lacunæ after absorption of the bone do not seem to play any active rôle in the pathological process.

The peculiarities of the vascular system constitute one of the characteristic features of osteogenic sarcoma. In the first place the richness in vessels of an osteogenic sarcoma is interesting. These neoplasms are far better supplied with blood channels than normal bone. The only exception is found in osteogenic

Fig. 39. Case 335. Showing metastases of osteogenic sarcoma to the skin; ossification about a hair follicle is seen.

sarcoma with a pure cartilaginous matrix as a base. The excess of vascularity may be seen also in the structures close to the tumor capsule where numerous, large blood vessels are seen. There was occasion to mention above the probable increase in malignancy of an extremely vascular osteogenic sarcoma. A telangiectatic osteogenic sarcoma is not more malignant because this is an essentially different disease but merely because abundant vascularity is a sign of lack of differentiation of the tumor growth, and as such it is a direct sign of greater malignancy. This suggestion is also supported by the fact that the blood vessels are the centers of very loose cellular areas (Fig. 28). The stage of differentiation is the higher the further the given area is from a blood vessel. This is also true of certain other skeletal dystrophies: sclerosis and ossification are usually at some distance from blood vessels. Calcification is apparently slowed down by the presence of blood vessels.

The blood vessels in osteogenic sarcoma are conspicuous in their anatomical structure and in their relationship to the tumor tissue. In the average pleomorphocellular osteogenic sarcoma numerous blood capillaries may be seen coursing along clefts between tumor cells. The tumor cells seem to carry the function of the endothelial lining of these blood channels. The same relation to the tumor

may be frequently noticed in blood vessels of larger caliber. Areas can be seen where cells of exactly the same morphological structure and appearance are lining blood vessels in one field and are indulging in bone formation in another. In still other places the endothelial lining of blood vessels seems normal but the wall consists of a homogeneous mass exactly like osteoid or near osteoid tissue (Figs. 29, 30). It is also not uncommon to find a cartilaginous matrix in place of the homogeneous mass forming the wall of the blood channel (Fig. 31). The lining of a capillary with tumor cells raises the question of the origin of such a capillary. If the capillaries here are formed according to general histological rules then they are formed by sprouting of a column of tumor cells, in other words there must be a most intimate relationship between normal endothelial cells and the tumor cells. These peculiarities of the blood vessels encountered in osteogenic sarcoma have been especially emphasized by Codman, who suggests for these the term "tumor blood vessels." Codman sees in these peculiarities a proof to his statement that the malignancy of bone sarcoma lies not in a certain kind of tumor cells but in the intimacy of the cells to the blood vessels. He suggests the idea that all osteogenic sarcomata are endotheliomata because, he thinks, the tumor cells spring from blood vessels as leaves from a tree. Whatever one's opinion may be, about Codman's broad statement, there is probably more truth than error in a belief that the malignant impulse imparted to the cells of a sarcoma lies much further back than in the origin of a "malignant set of cells." It may reside in a disorder of the laws of growth.

A peculiarity in the histology of osteogenic sarcoma which deserves special emphasis is a spontaneous lymphocytic infiltration (Fig. 32). A lymphocytic infiltration is not uncommon in tumors after radiation therapy. It is also frequent in osteogenic sarcoma in which an exploratory incision was made previously; it then being a simple inflammatory reaction to the infection brought in during the operation. A spontaneous lymphocytic infiltration however is not frequent; it is met with in about 5 per cent of all cases. That the lymphocytes are not an integral part of the tumor one can conclude from the fact that they are usually found on the periphery of the tumor. In the 17 cases with a five years' cure of primary malignant bone tumors registered, the majority of osteogenic sarcomata showed a marked lymphocytic infiltration, although several of these have had previous operations. This fact raises the question whether the lymphocytic infiltration is not an equivalent of the feature encountered in early carcinoma as pointed out by Paltauf. The latter called attention to the fact that in early carcinoma there may be found a lymphocytic infiltration of the surrounding connective tissue, which according to him is a result of the reaction of the organism to the carcinomatous involvement. The same feature may be evinced by early metastatic carcinomatous involvement of regional lymph nodes while in similar involvement of an advanced carcinoma this reactive process is

absent. "Unwillingly one gains an impression that the organism has lost its resistance and the carcinomatous cells gained the upper hand." Ewing suggested the advisability of a separation of all cases of osteogenic sarcoma with a lymphocytic infiltration from the general group. However, not all cases with a lymphocytic infiltration in the Registry gave a five years' cure.

The remarkably high malignancy of osteogenic sarcoma is due to a generalization of the tumor by way of metastatic deposits in other organs. No rules can be made as to the localization of the secondary tumor growths. They are known to occur in organs of the abdominal cavity, in the brain, in other bones and even in the skin; but the most frequent, the classical seat of a metastatic osteogenic sarcoma is the lungs. Death from the development of pulmonary metastases is the outcome in a very large majority of cases of osteogenic sarcoma. A metastatic involvement of the regional lymph nodes is rare. Frequently it is difficult to decide from the section alone whether the lymph node does show a metastatic involvement; occasionally an extensive hyalinization of the stroma of the enlarged lymph node is the only sign of a metastatic involvement (Fig. 33). The metastatic growth usually shows a tendency to turn to a less differentiated and more cellular structure than in the primary tumor. On the other hand in anaplastic metastases most complex pleomorphic tumors may become simple. Usually the cells of the secondary growth may reach all the various stages of differentiation as seen in the primary tumor and it is not unusual to find a metastatic growth with all the varieties of structure of an osteogenic sarcoma.

Pulmonary metastases have a tendency to grow slowly. Cases are known in which the haversian canals of the bone found in pulmonary metastases contained a structure strongly suggesting bone-marrow. A study of a lung involved metastatically in osteogenic sarcoma may show all the successive stages of the development of the metastatic growths. The first localization of the metastases is in the subpleural portion of the lung (Fig. 34). Here, right beneath the pleura, can be seen later the typical grayish-white rounded nodules of various size and of a consistency ranging from that of brain tissue to a solid, hard, ivory-like mass (Fig. 35). The tumor cells brought in the blood stream are retained in the interalveolar capillaries of the lungs (Fig. 36). In section one sees capillaries completely stuffed with tumor cells. Soon the tumor cells break through the wall of the capillary and begin to infiltrate the alveoli. The alveoli are seen packed with tumor cells (Fig. 37). Then the interalveolar capillaries become obliterated, and it is with difficulty that one makes out the alveolar texture. The whole area is converted into a tumor mass, with very faint traces of former alveolar structure of the lung. At a later stage bone may form throughout the mass (Fig. 38). A compensatory emphysematous dilatation of the adjoining alveoli completes the picture. Although uncommon, metastases to the skin with bone formation are known to occur (Fig. 39).

Clinical Course

A study of the clinical course of the primary malignant bone tumors has always attracted much less attention than the morbid pathology of these tumors. The explanation of this probably rests with the fact that the clinician was and still is helpless when confronted with primary malignant bone tumors. Even today the average clinician will gladly shift the task of diagnosis in such a condition to the roentgenologist and pathologist. A great deal of confusion in our knowledge of the clinical course was brought in by those who in their earnest attempts to give a description of the clinical course of these conditions have dealt with the primary malignant bone tumors as a whole, that is, as with a single clinical entity. Not less harm has been added by individual reports of single cases which erroneously were considered as "bone sarcoma." Clinical histories are not always properly taken in these conditions and the clinician does not pay sufficient attention to the history in the diagnosis. A complete, well taken clinical history very frequently can decide the question of the diagnosis in cases histologically obscure. Often from a good clinical history and roentgenograms one can be as sure of a diagnosis as from seeing the patient, the lesion, the gross specimen and numerous sections.

The incidence of primary malignant bone tumors is best seen from the fact that about one case out of three of sarcoma of the human body is a "bone sarcoma," the next in frequency being lymphosarcoma. From a careful survey in the State of Massachusetts, which, due to the influence of Dr. Codman, is the best educated in registering its cases of "bone sarcoma," it is estimated that the clinical incidence of "bone sarcoma" is about one to 100,000 of population at any one moment. According to recent estimates of English observers, there is in England about one case of bone sarcoma to 75,000 of population. Judging from the data of the Registry there occur about twice as many primary malignant bone tumors as benign giant cell tumors. According to these calculations there are about 1,100 to 1,450 patients with "bone sarcoma" alive at any one time in the United States. Allowing about one-third of this number for the benign giant cell tumor and considering the average life of a patient with a true malignant bone tumor to be 5 years, we have about thirty thousand deaths from sarcoma in a generation (60 years), deaths in individuals mostly in the prime of life.

Social conditions have no influence as an etiological factor and cases of this disease are found in all walks of life. Of no great influence also seems to be sex, although the male sex is predominating in osteogenic sarcoma, the relation being about 3:4. In the first decade, however, the female sex distinctly predominates in frequency.

Osteogenic sarcoma is primarily a disease of the young. The Registry material upon which the adjoining curve is based shows that while there is no age entirely

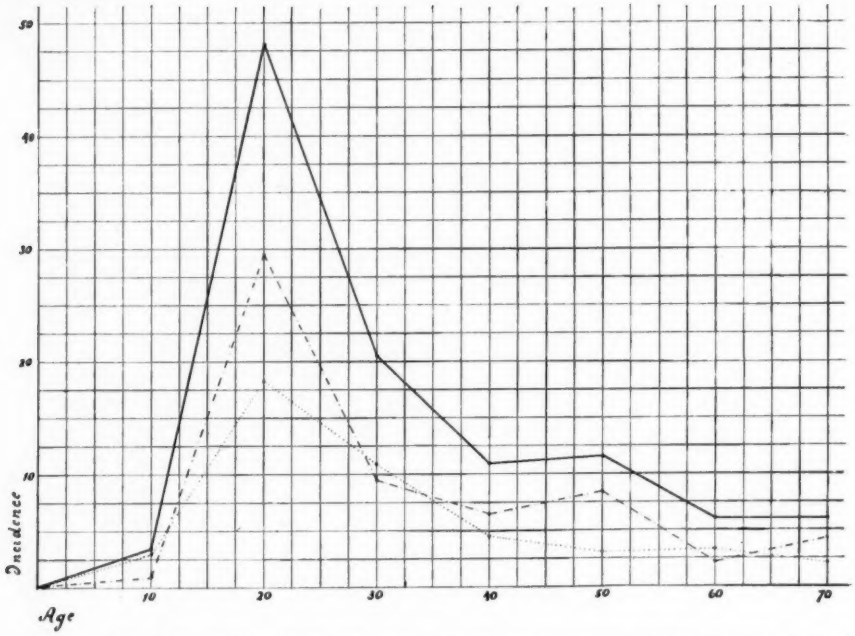


Fig. 40. Chart illustrating the incidence of osteogenic sarcoma in relation to age. Dotted line—females; broken line—males; heavy line—both sexes combined.

immune, the incidence of this tumor, however, predominates in the second decade of human life (Fig. 40). It is of the least frequency in the first decade, especially during the first few years of life. There is no authentic case on record of a congenital osteogenic sarcoma, the great majority of congenital sarcomata being located in the soft parts occasionally attached to the outer layer of the periosteum. The fact that osteogenic sarcoma develops most frequently during the period of the most energetic skeletal growth and the period of skeletal puberty has long been the basis for the inclusion theories of tumor origin and of theories of endocrine influences. The fact that the greatest frequency of osteogenic sarcoma is in the age of most active skeletal growth and development would certainly seem to indicate that energetic growth of the skeleton is one of the main factors in the etiology of these tumors.

While most bones of the skeleton may be the seat of osteogenic sarcoma they are, however, met especially frequently in the long bones. The metaphyses of the long bones are the seat of predilection of osteogenic sarcoma. This is especially marked in osteogenic sarcoma of the lower extremity which comprises about 72 per cent of all cases of osteogenic sarcoma; in 82 per cent of all osteogenic sarcomata of the lower extremity the tumor affects the region about the knee whether

it be the femur or the tibia. The relative frequency of osteogenic sarcoma in the upper and lower extremity may be expressed by the ratio 2:11; the tumors of the upper extremity comprising about 10.5 per cent of all cases of osteogenic sarcoma, and the relation between the extremities on the right and left side by the ratio 5:4. The individual bones of the skeleton with the highest frequency of involvement are the femur, tibia, humerus, all bones of the pelvis combined, the fibula, the bones of the shoulder girdle, the ulna, the bones of the hands and feet (exclusive of the phalanges of fingers and toes), the ribs, the skull, the jaws and the vertebræ. While the lower end of the radius is a common seat of giant cell tumor no authentic case of osteogenic sarcoma of this bone is known to the Registry, only two doubtful cases of osteogenic sarcoma having been recorded. The same is true of the lower end of the tibia, an homologous bone.

The bone most frequently involved in osteogenic sarcoma, the femur with its 52 per cent of all cases, has its seat of predilection in the lower metaphysis and in 82 per cent of all cases in the femur the tumor is situated in this region. The remaining 18 per cent are distributed in the shaft, about the great trochanter and in the neck. It is important to remember that about 9 per cent of all cases in the femur are located in the shaft of the bone without any gross involvement of either end. This is especially interesting since it is usually considered that an involvement of the shaft is pathognomonic of Ewing's sarcoma, and in several of the cases of involvement of the shaft in the Registry material this diagnosis has actually been suggested on account of this situation of the tumor. The features of this variety of osteogenic sarcoma will be pointed out more explicitly in the following chapter. The next seat in freqeuncy in the femur is the region about the great trochanter. Osteogenic sarcomata here are marked by the prevalence of cartilage. Degenerative changes frequently lead to cyst formation in these tumors. Occasionally the tumor appears to be attached to the trochanter by a well defined pedicle. The neck of the femur and the region close to the head are the seats of least frequency.

The second most frequently involved bone, the tibia, averages about 20 per cent of all cases. About 90 per cent of all cases situated in the tibia involve the region about the upper end of the bone. It is interesting to note that here the tumor is seen mostly on the internal aspect of the bone and very rarely on the lateral aspect. The remaining cases of osteogenic sarcoma are situated all along the bone except at the lower end.

The humerus carries about 9 per cent of all cases of osteogenic sarcoma, the majority being found in the upper third of the bone, very frequently about the insertion of the deltoid, while an osteogenic sarcoma below the deltoid tubercle is a curiosity. In the pelvis with its 5 per cent of all cases of osteogenic sarcoma the tumors are most frequently situated in the ilium, usually about the crest; sometimes they are found in the os ischium and rarely in the pubis. It is of

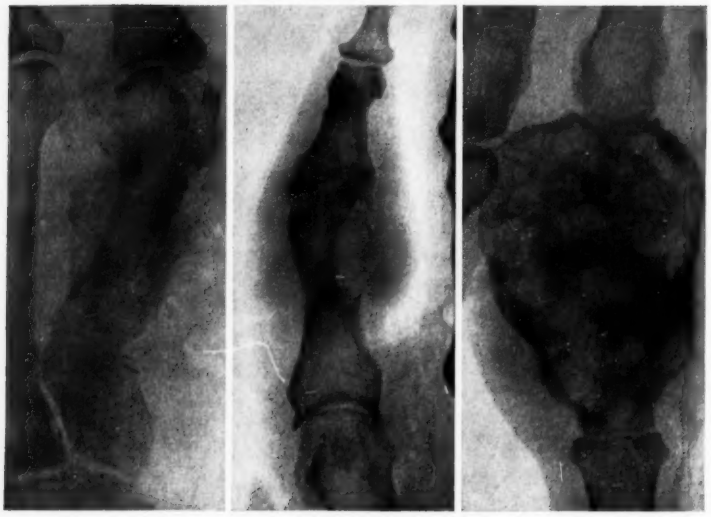


Plate 8. Roentgenograms showing (1, left) osteogenic sarcoma in a young woman; (2, center) solitary gout nodule which simulates an extraperiosteal sarcoma with pressure absorption of bone; and (3, right) enchondroma.

interest that the tuber ischii which is under frequent physiological stress is an uncommon seat of osteogenic sarcoma. The fibula is usually involved at its upper end; localization of an osteogenic sarcoma in the lower end of the fibula is rare. Of the bones of the shoulder girdle, the scapula is the seat of predilection, especially about the glenoid fossa. The ulna is usually affected at its proximal end, no case of osteogenic sarcoma in the distal end having been registered. Only the large bones of the feet are occasionally affected by osteogenic sarcoma; the phalanges of the fingers and toes are free from true osteogenic sarcoma (Plate 8). In fact no case of metastases from an osteogenic sarcoma of the phalanges is known. The skull is affected by osteogenic sarcoma mostly in children. An involvement of the lower jaw is more frequent than that of the upper. The vertebræ are very rarely the seat of an osteogenic sarcoma. An osteogenic sarcoma of the patella has never been registered.

The fact that a great majority of osteogenic sarcomata are found in the bones of the lower extremity led to a hypothesis that physiological stress and strain are in some way a predisposing factor in the etiology of osteogenic sarcoma. While it is true that the lower extremity is under incomparably higher stress and strain than the upper extremity, it cannot be denied that the spine far exceeds the lower extremity in this respect, yet osteogenic sarcoma of the spine is not common. On the other hand the localization of most osteogenic sarcomata about the knee, which is always exposed to trauma, suggests that trauma is a definite etiological

factor in osteogenic sarcoma. It has been mentioned above that the fact that the majority of osteogenic sarcomata occur in the young suggests that energetic skeletal growth plays a certain rôle in the etiology of osteogenic sarcoma. This suggestion is well in accord with the fact that the lower epiphyseal end of the femur and the upper epiphyseal end of the tibia are the last to ossify in the human being, remaining in the growing age until the first years of the third decade.

Osteogenic sarcoma is a solitary disease. In the literature where, under the heading of "bone sarcoma," are embraced together with osteogenic sarcoma also Ewing's sarcoma and myeloma, multiple bone sarcoma is frequently mentioned. It is of course not uncommon to find a multiple Ewing's sarcoma. In myeloma multiplicity is the rule. That myeloma has frequently been described as multiple "bone sarcoma" may be suspected from the fact that most reported multiple bone sarcomata were medullary in location. It would not be surprising if under certain conditions favorable for osteogenic sarcoma a patient should develop simultaneously more than one lesion; yet multiple cases are not common. It is impossible to decide whether a rare multiple osteogenic sarcoma is such from the start or whether one tumor is the primary growth and the others are of metastatic origin.

Sometimes an osteogenic sarcoma is superimposed on multiple chondral exostoses, in which case multiple osteogenic sarcomata may be encountered. Multiple lesions are also known to occur in patients with Paget's disease (osteitis deformans). Such cases often pass as metastatic from a single primary osteogenic sarcoma. Codman pointed out the fact that because most cases of osteogenic sarcoma occur under the age of 50 and most instances of Paget's disease are recognized over the age of 50 the presence of an osteogenic sarcoma in later life is almost presumptive of Paget's disease. The Registry material shows that in about 5 per cent of all cases of osteogenic sarcoma the tumor arises in Paget's disease. Codman believes that about 14 per cent of all cases of Paget's disease succumb to osteogenic sarcoma. It has been suggested that osteogenic sarcomata occurring in Paget's disease be separated as a definite entity. There is, however, no clinical evidence to support such a separation. As in most cases of osteogenic sarcoma, the etiological factors of osteogenic sarcoma in Paget's disease probably may be explained by local loss of growth restraint (repair restraint). Such a local loss of growth restraint when repair is called for has serious consequences in Paget's disease with its apparently constitutional weakness of general growth restraint (Plate 9).

In the clinical history of a patient with a primary malignant bone tumor there are two features which attract the attention of the diagnostician. One of these elements is pain, which is the first symptom in a patient with an osteogenic sarcoma. It precedes the appearance of a tumefaction by an interval lasting a

Plate 9. Courtesy of Dr. Holmes, Massachusetts General Hospital. Osteogenic sarcoma in a man of 49, with Paget's disease. The patient sustained a fracture of the femoral neck. While immobilized in a hip splint, he developed an osteogenic sarcoma. Roentgenograms taken soon after the fracture and 3 and 5 months later.

few days, weeks, or months. This symptom is of such importance that severe persistent pain in a long bone of a young adult should cause a suspicion of sarcoma. Pain is due to the high sensitiveness of the periosteum which invariably becomes affected to a greater or lesser degree in malignant bone tumors. Sometimes, however, pain is not proportionate to the seriousness of the condition; all the patient experiences first is a tired feeling, some uncertainty of the muscles about the adjoining joint and hence limping. When the process encroaches upon the periosteum, pain appears. Pain is of a very severe, boring, excruciating or rheumatoid and gnawing character. The suffering is especially agonizing and unbearable during the night, waking the patient from sleep. In fact the sleepless nights are the main cause of the run-down general condition so frequently seen in patients with osteogenic sarcoma. In the early stages pain may be intermittent in character. Pain of a rather dull character comes on immediately after trauma, lasts one to two weeks, disappears and all that is left is perhaps a slight limp. Some time may pass and the pain reappears, but of a deep seated excruciating character. Occasionally a new trauma precedes the reappearance of pain. Intermittent pain, however, is more typical of a Ewing's sarcoma than of osteogenic sarcoma.

The best explanation of the cause of pain is pressure of the tumor mass from within the bone upon the periosteum. That tension of the periosteum, and not pressure upon nerves as suggested by others, seems to be the cause may be concluded from the fact that in far advanced osteogenic sarcoma after the periosteum is perforated by the growing tumor mass, pain may disappear; the patient is up and around until a pathological fracture or metastases oblige him to become bed ridden. Pain is also relieved by drawing up the leg so as to relax the muscles overlying the tense periosteum. The spectacular and almost immediate disappearance of pain after radiation could hardly be explained by relief of pressure upon nerves, but it is well in accord with the radical influence of radiation upon the rapidly growing tumor. There are other conditions of bone in which pain is of similar severity, e.g., acute osteomyelitis with an accumulation of pus between the cortex and the periosteum. The pressure of the pus under high tension upon the periosteum causes the pain, and drainage here immediately relieves it. Similar pain is observed in syphilitic periostitis and in sclerosing non-suppurative osteomyelitis—conditions which greatly affect the periosteum. The appearance of pain is not in any way to be identified with the onset of the tumor. The not infrequent short duration of life after the onset of pain is the best proof that the tumor was present long before, and only when the periosteum became distended to a certain point did the pain call the patient's attention to the condition.

Another important element in the clinical history is trauma. The question of the importance of trauma as a diagnostic point in "bone sarcoma" has been

debated for years. As our knowledge stands today, one may say that trauma is an unreliable element in the history of a patient with a primary malignant bone tumor, and this is as true of osteogenic sarcoma, as of Ewing's sarcoma. The fact that statistical data vary greatly in the estimation of trauma indicates that there must be important sources of error in such statistics. The main error is evidently the question about which an immense confusion exists: "What is to be considered a trauma?" Is not a sudden tension of a muscle upon the bone to which it is inserted also to be considered a trauma? In accordance with the answer to this question the frequency of trauma in the clinical history will obviously change. Another error is due to the fact that the history of trauma is purely subjective and is apt to lack in trustworthiness; it is an old inclination of a patient to connect up his suffering with some external fact as a cause. We all frequently suffer traumata which usually pass unnoticed, but in the exceptional case when a tumor develops, we recall the trauma history. In some cases a trauma is mentioned by the patient for purely mercenary reasons. There also can be no doubt that in many cases the quasi-responsible trauma was sustained after the actual onset of the tumor. This fact is not sufficiently realized even by the diagnostician; meanwhile the occurrence of pulmonary metastases and death after a very short clinical course, 2 to 3 months after the discovery of the tumor by the patient, strongly support the possibility that the trauma in the patient's history took place after the actual onset of the tumor.

While there can be no doubt that the old motto, "When there is no trauma there is no tumor" is obsolete and not in accord with actual facts, there is, however, strong evidence to support the view that in general trauma may lead to osteogenic sarcoma under certain predisposing conditions. It is probable that trauma is the exciting cause of all osteogenic sarcomata but it seems that the particular trauma associated with the onset by the patient is seldom a causal trauma. The strongest argument against the importance of trauma in primary malignant bone tumors is the fact that osteogenic sarcomata are rare after fractures. However, sarcoma in callus after fractures does occur, to be sure not as frequently as one might expect if this kind of trauma were important in the etiology of osteogenic sarcoma (Fig. 41). As a matter of fact, the character of trauma preceding osteogenic sarcoma is usually not a severe blow producing a fracture, but a more or less slight trauma of the contusion type, frequently forgotten by the patient until reminded of it by the appearance of pain. In this all sarcomata differ from carcinomata where not a single but a repeated, more chronic trauma is important. Slight repeated traumatizing, however, is not infrequent in the histories of cases of osteogenic sarcoma. The trauma here is of a chronic irritative type, one which can hardly be qualified as a trauma in the usual sense of this word, for example an osteogenic sarcoma which arises in a bone scar of a cured osteomyelitis. Typical in this country are osteogenic

Fig. 41. Case 156. Osteogenic sarcoma in a boy 14 years old. The tumor arose at the point of a well united fracture which preceded the clinical onset of the tumor by 7 months.

sarcomata appearing after a sport trauma—football or baseball. In such cases the tumor is occasionally accompanied by myositis ossificans.

The question of how a trauma may lead to an osteogenic sarcoma furnishes ample material for speculation, most alluring being the theory of loss of repair restraint in the bone after the trauma. Important from a practical standpoint is the fact that the history of trauma is very frequently obtained from a patient with an osteogenic sarcoma and hence trauma plays a definite rôle as a diagnostic point in this disease. A history of trauma is more frequent in patients between the ages of 10 and 30, perhaps because they are more active and less careful than older people.

Of obvious importance is the time elapsing between the sustained trauma and the appearance of the tumor. In a large series of cases one finds the most discrepant intervals of time ranging between a few days and a year or more. The very short intervals of time are evidently of no diagnostic value; also the longer the intervals the less reliable is the history of the trauma. The most frequent interval is about one month between the time of the trauma and the appearance of pain or tumefaction. This interval is actually found in approximately one-third of all cases of osteogenic sarcoma.

Unlike carcinoma the general condition in malignant tumors of bone is generally good until the later stages of the disease. Of course night pains keep the patient in misery and may cause a rapid running down of his general condition, but as soon as this suffering is relieved spontaneously, by radiation, or amputation, the general condition of the patient is again satisfactory. Occasionally one observes a case of osteogenic sarcoma in which the temperature is high. The febrile stage usually lasts but a few days, disappears, and may recur at a later date. Such a febrile course, however, is more common in Ewing's sarcoma than in osteogenic sarcoma. When the patient enters the last stage—the generalization of the disease—a febrile stage is frequent, especially in the presence of pulmonary metastases with an accompanying consolidation of the entire lung. Along with the high temperature one sometimes finds hyperleucocytosis with a predominance of lymphocytes. Myelocytes are not a constant finding; when present they comprise about 3 to 5 per cent of the leucocytes. The clinical reaction of the patient to the disease in the terminal stage is also unlike carcinoma. Instead of cachexia, one encounters here a grave anæmia. The chalk pale patient has a characteristic haggard appearance, which when once seen is never forgotten. Occasionally the persistent lowering of the hæmoglobin, in spite of a good general condition of the patient, is a sign of a generalization of the disease. When the tumor is infected after an exploration, the general condition of the patient rapidly changes for the worse and may be followed by an appearance reminding one of cachexia.

The reaction of the skin overlying the affected region is also unlike that which is usually observed in carcinoma. While in carcinoma the skin overlying the

tumor early becomes involved, in osteogenic sarcoma the skin is persistently stretched by the tumor, but not invaded by it. It preserves its mobility and natural color for a long time. However, frequently the temperature of the skin overlying the involved area is distinctly higher than that of the neighboring areas. Early, dilated veins become noticeable. These dilated blood channels in the pale white skin give a striking danger signal to the observer. The skin usually remains intact, and if no exploration is done, an ulceration of the skin is rare. Occasionally, however, the skin becomes œdematous; it is tense and glossy, the superficial veins are very large. When the tumor is far advanced and the thinned out skin looks inflamed due to a dilatation of the superficial veins it acquires a purplish tint which may be followed by extensive excoriation. Inflammation may even go on to ulceration although this is very uncommon. These changes in the skin are of little diagnostic importance because they are observed late in the course of the disease.

The shape, outlines, and the consistency of the tumor are well perceived through palpation, and it is important to repeat such examinations frequently. By palpation one may often outline the anatomic borders of the tumor with a surprising exactness. Repeated palpation before and after radiation may bring out even the slightest changes in the consistency, size, and shape of the tumor, frequently most significant in establishing the diagnosis. In tumors with a very thin periosteal investing capsule and cystic degeneration one may encounter crackling; it is, however, a very uncommon finding. In very vascular tumors a pulsation and bruit can be felt. In the following chapter this feature will be dealt with in greater detail. Palpation may sometimes discover to the patient tenderness of which he was unaware before, since tenderness may be present in the absence of pain. It goes without saying that only when the utmost caution is exercised in palpation is the latter permissible.

The size of an osteogenic sarcoma is variable. In general in these days large malignant bone tumors are less frequent than twenty-five years ago—the tumor is usually diagnosed much earlier. Much depends on the seat of the tumor. When in close proximity to important structures an increase in size of the tumor may affect them and in this way draw the patient's attention to the disease. Usually only the primarily benign but potentially malignant skeletal chondromata of long duration reach a large size, before they become actual osteogenic sarcomata. The rapid and extensive regressive changes in malignant bone tumors may considerably slow down the increase in the size of the tumor. It is unsafe to attempt to state the time of onset of the tumor from its size. The rapidity of growth and the increase in size depends greatly on the consistency of the tumor tissue; the firmer osteoblastic tumors are of more moderate dimensions than the soft encephaloid variety. As a rule after the tumor breaks through the bone and the investing periosteal capsule, its rate of growth markedly increases. During

Fig. 42. Case 469. Osteogenic sarcoma in a boy 15 years old. Showing an extension of the tumor into the knee joint.

the course of an osteogenic sarcoma traumatism of any kind may lead to an acceleration of growth. By traumatism is meant here local direct as well as indirect general traumatism, for example hard physical labor, exercises of any kind, an exploratory incision or puncture of the tumor. After an exploratory incision the acceleration of growth is usually very acute. The fact that hard physical labor may have a sudden accelerating effect on the tumor may be important in the courts. The rate of tumor growth may be and probably always is accelerated by a pathological fracture through the involved area of bone.

In general, pathological fractures are not typical of osteogenic sarcomata. Usually tumefaction and pain are keeping the patient off his feet. They are more apt to occur in osteogenic sarcomata growing expansively in all directions in the involved bone, in the so-called central variety. The fracture follows a trifling trauma or force. It may also result entirely spontaneously and sometimes even without the patient's knowledge of it. Pathological fractures are usually transverse, not "green stick" but rather "rotten wood" fractures, although oblique pathological fractures do occur. Like pathological fractures in carcinomatous involvement healing of fractures here is not uncommon. Instead of callus it may unite by means of ossifying tumor tissue with a subsequent refracture when the new formed bone is destroyed by more cellular tumor tissue.

The reaction of the joint close to the involved bone end in malignant bone tumors is peculiar. It is interesting to note that a patient complains less of

Fig. 43. Case 316. Skeletal chondroma in a man 58 years old. Onset in 1913. Local excisions of the tumor and recurrences in 1915, 1920, and 1923. The histology of the last recurrence showed distinct malignant features. The present roentgenogram was taken before the last operation.

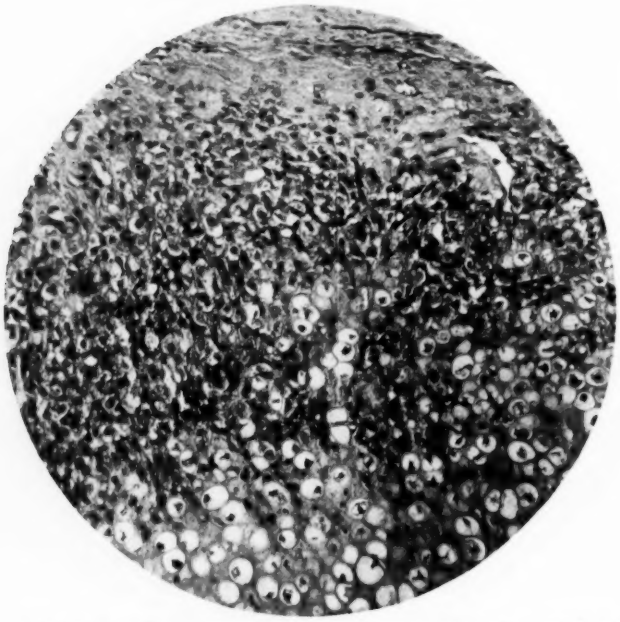


Fig. 44. Case 296. Same case as in Figures 45, 46, and 47. Skeletal chondroma. Showing a characteristic conglomeration of various typed cells beneath the fibrous capsule of the tumor.

articular pain in osteogenic sarcoma than in osteomyelitis. There may be initial soreness and perhaps an effusion into the joint cavity following trauma, but after the exudate absorbs, no articular discomfort is felt by the patient. Movements in the joint are free. In the far advanced stages of an osteogenic sarcoma there may follow a sarcomatous involvement of the neighboring joint. While involvements of the joint by extension from the tumor do not occur very frequently, they are, however, in my experience not uncommon. Because the articular cartilage is very resistant to sarcomatous involvement a penetration of the tumor into the joint does not take place through the articular cartilage (Fig. 42). It usually takes place following a pathological fracture through the involved end of the bone. Such joint involvement is more frequent in the adult than in the young, because of the completed fusion of the epiphysis; in the former the tumor frequently spreads directly in the epiphysis. When the joint is involved, it sometimes becomes distended with fluid and shows signs of fluctuation; this is, however, the exception and not the rule. Usually a fusiform enlargement of the joint slightly resembling a tuberculous arthritis is seen. Cases are even known of a diagnosis of tuberculosis on account of this similarity. On opening the joint the synovial membrane is found converted into a grayish red fungous tumor mass

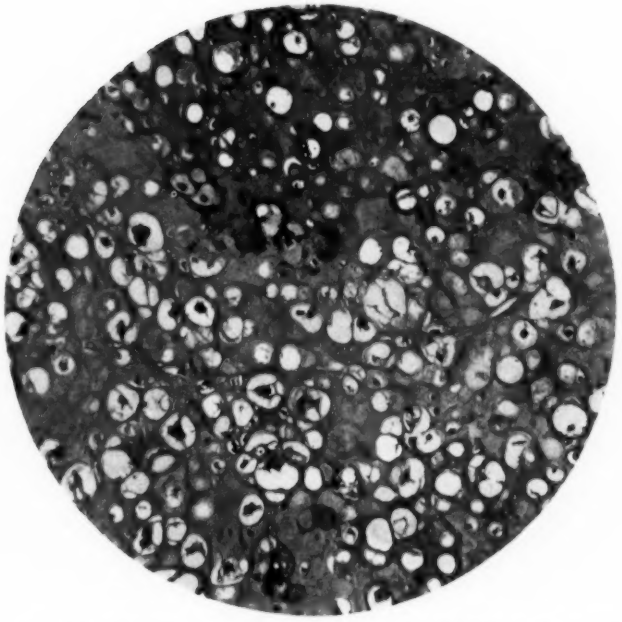


Fig. 45. Case 296. Compare with Figures 44, 46, and 47. Showing the preponderating structure of the tumor—atypical cartilage.

which is in direct communication with the extra-articular tumor. Sometimes the synovia is converted into a large nodular encephaloid mass. The knee is the joint most frequently involved; but other large joints may be affected in a similar way. There is some evidence that an osteogenic sarcoma after perforating into the joint may involve the adjoining normal bone from within the joint cavity. This is, however, very unusual.

One of the most constant manifestations of the clinical course of primary malignant bone tumors is generalization of the tumor, through metastatic dislodgment. In the literature one frequently finds most definite statements as to the frequency of metastases in malignant bone tumors. Similar statistical data are of no value unless they are based on cases histologically proved to be osteogenic sarcoma with the presence of metastases well established. In primary malignant bone tumors all similar statistical data are of relative importance because of the exceedingly grave prognosis of the disease; in only exceptional cases does the patient live more than five years, while in the majority of cases death is a result of generalization.

The idea that the blood stream is the only path of spreading of metastases in sarcoma does not hold true in malignant tumors of bone. The lymphatic system

Fig. 46. Case 296. Compare with Figures 44, 45, 47. Focal hyalinization with ossification of the tumor.

occasionally plays a definite rôle in the transportation of tumor tissue in the organism. The importance of the blood channels in the generalization of the disease is, however, predominant. A perforation of the tumor mass into a blood channel is not uncommon, because of the intimate relation of tumor elements to the blood vessels as pointed out above. As a result of venous transportation of the tumor cells, pulmonary metastases are most frequent. From the lungs a dissemination may follow through the skeleton and parenchymatous organs. The perforation of a pulmonary metastatic mass into a bronchus with expectoration by the patient of fragments of seminecrotic tumor tissue has come under my personal observation. The incipient clinical signs of a pulmonary metastatic involvement may be the same as those in diffuse bronchitis. Sometimes a patch of pneumonia is diagnosed, the patient showing all the symptoms and signs of it—cough, dullness, fever, leucocytosis. In this connection it is important to remember that in cases in which, on physical examination, no involvement of the lung is found, a radiological examination may reveal early nodular metastases. Sometimes one is confronted with the necessity of differentiating in the roentgenogram between healed tuberculosis and tumor metastases. The best point of differentiation is the fact that in healed tuberculosis the nodules throw a deep, more or

Fig. 47. Case 296. Compare with Figures 44, 45, and 46. Showing an area of myxomatous degeneration of the tumor.

less rounded shadow the size of a large pea; this is due to the calcification taking place in healed tubercles. In early sarcomatous metastases no bone production is present usually and the shadow in the roentgenogram is faintly outlined. Negative physical and radiological findings do not rule out an early pulmonary involvement. Typical sarcomatous metastases are globular in shape, few in number, and large in size.

In the absence of pulmonary metastases, metastatic tumors have been found in other organs, especially in the bones. Some of these may be explained by retrograde transportation of the dislodged tumor elements, through veins, to their new location. In some the absence of pulmonary metastases is probably more apparent than real; it is also known that radiological evidence of pulmonary metastases may entirely disappear temporarily after radiation therapy.

Spreading of tumor metastases through lymphatics is infrequent. It is obvious that a metastatic involvement of regional lymph nodes in malignant bone tumors means more as an indication of generalization than in carcinoma. An occasional simple lymphadenitis in malignant bone tumors should be clearly distinguished from a metastatic involvement of the nodes. Such a lymphadenitis is usually found following an infection after ulceration of the tumor or after an exploratory

incision. However, an inflammatory reaction in the regional lymph nodes is also encountered when there is no ulceration present.

Naturally, a question arises as to the interval of time between the onset of osteogenic sarcoma and the appearance of metastases. No definite answers as to this question can be made; in certain cases pulmonary metastases and death have been known to take place two months after the clinical onset, in others metastases have not been discovered until years after the onset. Usually metastases develop during the first two years after the onset. Cases are known in which pulmonary metastases led to the discovery of the primary tumor. In this respect osteogenic sarcoma is like carcinoma—not infrequently extensive skeletal carcinosis may be the first indication of the presence elsewhere of a primary tumor.

Skeletal chondroma is related clinically to osteogenic sarcoma. This is a primarily benign tumor which is notoriously persistent, grows to a large size, tends to recur after incomplete surgical procedures and finally to metastasize. The most frequently given history is that for some cause a tumor developed. It reached a considerable size; it remained benign for years until some kind of trauma, accident or a surgical operation, led to a sudden increase in vitality, to a distinctly malignant change, and to metastases and death. Putting it in terms of the repair theory the trauma calls for a new attempt at repair in the tumor; but because growth restraint is low or entirely absent, as is suggested by the primary development of the tumor, a more malignant tumor develops. The long space of time before a sudden turn for the worse after the trauma is the prevailing feature of this type of tumor. At operation the tumor appears perhaps not to involve the cortex of the bone on top of which it is situated, a local excision is done, and a rapid recurrence follows. Because of recurrence repeated local operations are performed. These surgical operations are followed by an increase in the blood supply of the tumor and by a change to malignancy. Their metastases and final course are typical of osteogenic sarcoma. These tumors are usually located in the diaphysis near the epiphyseal end of the bone. Sometimes they are distinctly pedunculated, in which case they are of far better course and prognosis. Frequently, however, the pedunculation is more apparent than real. They may lie entirely beneath the periosteum, projecting externally and pushing outward the periosteum; they may simultaneously grow in the medullary cavity, distend the latter and become surrounded by a thin bony shell. When they extend far, they may prove malignant from pure mechanical factors. The most frequent location of these tumors is the lower end of the femur, about the popliteal space (Fig. 43). Here they may reach an enormous size without great discomfort to the patient. They are frequent about the hip joint, especially about the great trochanter. The insertion of the broad pedicle to the trochanter is noted. In this location the tumors often pass for fascia sarcoma. Similar tumors are known to occur also in the upper end of the humerus and in the neck

of the scapula, about the glenoid fossa. Occasional instances of this tumor occur in various other locations. The histological structure of skeletal chondroma is manifold; it frequently resembles an osteogenic sarcoma with a preponderance of a cartilaginous matrix (Figs. 44 to 47).

Diagnosis

There are few chapters in medicine where exact diagnosis is hampered by so many difficulties as in malignant bone tumors. The confusion in the nomenclature and classification together with the fact that commonly these tumors are seen only at one or another stage of development, and the peculiarly complicated reactive processes of bone which contribute to the wide variations in their histological structure render the primary malignant bone tumors a complex diagnostic problem. Here it is that a collective study of many individual cases may greatly clarify diagnostic points. A very valuable contribution on the diagnosis of osteogenic sarcoma is written by Codman.[1] The diagnosis of malignant tumors of bone rests upon a careful consideration of the findings accumulated from all three angles of study: the clinical, roentgenological, and pathological.

Occasionally the history and clinical picture of the condition will suffice to establish the diagnosis, without the necessity of roentgenological or microscopical study; not infrequently a roentgenogram is more decisive in the diagnosis than a number of microscopical sections, but it is not at all uncommon for a most careful scrutiny and perusal of all available clinical and laboratory methods to prove insufficient for establishing a diagnosis. In general it is fundamentally wrong to emphasize the importance over the others of any one of the three angles of study, clinical, roentgenological, or pathological.

The importance of the history and the clinical course of a tumor of bone from the diagnostic standpoint has already been pointed out. These data are especially valuable in the early stage of disease when no tumefaction can be demonstrated. It is in this early stage that the patient's complaints are usually diagnosed as articular rheumatism. Articular rheumatism is the *primum refugium* of the unscientific physician not only in malignant skeletal tumors but in various other pathological conditions of the skeleton. It is indeed surprising how frequently this diagnosis is made by the physician in the presence of symptoms which, if evaluated intelligently, would lead one away from this diagnosis. Freedom of movement of the joint is an important differentiating feature between articular rheumatism and osteomyelitis on one side and osteogenic sarcoma on the other. In the latter, complete or relative freedom of motion in the adjoining joint is the rule except in occasional, far advanced cases when a secondary sarcomatous involvement of the joint proper takes place. Repeated careful palpation will not infrequently lead to an early discovery of a tumefaction.

[1] Codman, E. A. Registry of bone sarcoma. Surg., Gynec. & Obst., 1926, xlii, 381.

A clinical finding which is frequently misinterpreted and therefore misleading in the diagnosis is pulsation of the tumor. Allusion is made here to pulsation resident within the tumor itself and not to pulsation transferred to it from contiguous arteries. The occasional observation of pulsation and bruit in bone tumors has led to the diagnosis of these tumors as aneurysm of bone. One of the causes of pulsation in the tumor is richness in blood supply. In cellular, rapidly growing tumors extensive regressive changes may be followed by a formation of blood cysts and spaces which may cause pulsation. However, in skeletal tumors these factors cannot explain pulsation satisfactorily. The difference in the pulsation of a soft tissue tumor and a skeletal tumor may be brought out by a compression of the afferent main artery when the compression is followed by the disappearance of pulsation in a soft tissue tumor but not the entire disappearance in a pulsating skeletal tumor. This is probably due to the fact that the bone-marrow is pulsating normally. Apparently the influx and reflux of blood in the wide-meshed and thin-walled abundant capillaries transmits the pulsation to the delicate and loose cellular tissue of the bone-marrow. Whatever the exact nature of this tumor pulsation may be, it is fully attested that no aneurysmatic skeletal tumors exist.

To read and interpret correctly a roentgenogram of a bone tumor is an art acquired only with wide experience, combined with a thorough knowledge of gross pathology. For this reason individual statistics showing the high or low percentage of cases diagnosed correctly from the roentgenogram are not of much importance. The tendency in vogue a decade ago to overestimate the importance of a roentgenogram in the diagnosis of a skeletal tumor has been followed lately by a swing toward underestimating its importance. This swing has taken place because of the frequent errors in the diagnosis if one depends on the roentgenogram alone. The numerous difficulties which render a roentgenological study of an osteogenic sarcoma a complex problem are evident. The roentgenological features of an osteogenic sarcoma greatly depend upon the stage of the clinical course and upon the structural peculiarities in the tumor. The extensiveness of the tumor whether more medullary or cortical, the histological structure of the tumor, the destructive and proliferative bone reaction, the regressive changes with occasional subsequent formation of blood spaces and sinuses and cysts—these are just a few of the factors strongly influencing and complicating the roentgenological features of osteogenic sarcoma.

The great difficulties connected with the establishment of a correct diagnosis of osteogenic sarcoma from the roentgenogram, however, do not reduce its proper importance as a diagnostic aid. With our growing knowledge of this subject it is easy to see that in the future we shall have to make more extensive use of the roentgenological features of bone tumors for diagnostic purposes. From a study of the extensive material of the Registry one arrives at the conclusion that roentgenology is not sufficiently appreciated by the average physician in dealing with

bone tumors and too few roentgenograms are being taken. Not only among general practitioners and occasional surgeons but also in special surgical institutions the fact is not sufficiently realized that a roentgenogram is to be looked at as a method, harmless for the patient and most important for the physician, of frequent re-examination of a patient with a skeletal malignant tumor. In the presence of a suspected skeletal malignancy most of the bones of the skeleton should be examined roentgenologically. This is the only way to reduce *ad minimum* the number of cases in which a multiple tumor is to be mistaken for a solitary one. From the numerous unsatisfactory roentgenograms one sees how frequently most important technical points of roentgenology are not appreciated and are neglected. Probably the best films are secured with no screens and with long exposure. Because distortion is increased toward the margin of a film no details can be seen at the marginal zone of a large film which would warrant a diagnosis of importance. Such roentgenograms, to present details, must be taken with the proper tube and film—fine focused tube, small cone, small film. The Bucky diaphragm requires especial care since the lead striations showing up on a film in faulty technique cloud the picture and frequently prevent the correct analysis of it. Films at the usual two planes intersecting at right angles are indispensable here. Since a certain amount of detail is constantly lost in a print it is obvious that when important conclusions are to be drawn from the roentgenogram all studies ought to be made from the negative.

Speaking in general terms one may mention the following features of an osteogenic sarcoma which are frequently sufficient to differentiate it from other skeletal lesions. One of these features is the common absence of a definitely limited outline of the sarcomatous tumor. Since osteogenic sarcoma frequently spreads and for some time remains beneath its periosteal investing capsule a spindle shape of the tumor is common. In rapidly growing tumors the fusiform swelling is ossified only at the ends where the periosteal reaction succeeded in producing a bone shell in the investing capsule. In such cases the tumor will be revealed in the roentgenogram by a wedge shaped osteophyte at each pole of the spindle, springing from the periosteum and slowly fading away in the unossified soft tumor. The wedge shaped osteophyte is frequently spoken of as lipping of the periosteum. However, no one of the three mentioned features is absolute proof of malignancy, non-sarcomatous skeletal lesions sometimes presenting one or another of these features. This makes the diagnostic value of these points of only relative importance. In the presence of strong clinical evidence of malignancy roentgenological findings negative for malignancy are of little value.

The roentgenological appearance of an osteogenic sarcoma depends greatly upon the character of the bone reaction of the tumor and its degree of differentiation. It is chiefly roentgenologically that osteogenic sarcomata may be divided into osteoblastic and osteolytic. Taking general features, two types of ossification

can be distinguished in osteogenic sarcoma. One is the structural dense bone which springs from or is attached to the involved old bone, appearing on the roentgenogram as spicules radiating from the bone or as thin layers of osseous tissue parallel to the bone. The other type of ossification is structureless. The roentgen-ray shadow is spotted due to the presence of opaque areas in the tumor. It is commonly believed by radiologists that islands of opacity in a large tumor indicate that it is benign, since these areas are thought to be due to calcification and not true ossification. This belief is erroneous; frequently irregular so-called low grade bone will cast such a spotted, speckled shadow.

The most widely known roentgen-ray appearance of an osteogenic sarcoma is the so-called fan-like, sun-ray structure. The histology of these spicules and the probable cause of this arrangement has been mentioned above. This sun-ray arrangement of the new formed bone has for long been thought absolutely pathognomonic of osteogenic sarcoma. A similar arrangement in other skeletal lesions —in chronic inflammatory processes—is known to occur. Only in 18 per cent of the cases of osteogenic sarcoma of the Registry material a sun-ray arrangement of the new formed bone is seen. On the other hand cases of osteogenic sarcoma have been observed in which the arrangement of the new formed bone was in longitudinal striations. Such longitudinal striations, rare in osteogenic sarcoma, are usually observed in cases with a wide involvement of the shaft of the bone, similar in situation to that usual in Ewing's sarcoma.

There has been expressed an opinion in the literature that the arrangement of new formed bone in an ossifying tumor is chiefly dependent on its amount, on the rate of formation, and not on the nature of the cells from which it springs. These authors maintain that if the rate of growth of the tumor is slow and ossification tendencies are great, the newly formed bone is usually deposited with a spongy arrangement and not in radiating spicules. These statements seem purely contentious and unavailing in the face of the evidence accumulated from an examination of the Registry material with this point in view. If all cases of osteogenic sarcoma of the Registry are divided in two groups, in one group all cases before the twentieth year of age and in the other all patients above twenty years old; the first group—when the growth of the skeleton is at its height—shows that bone production is predominant in 86 per cent of all cases, while in the second group—when the skeletal growth has been completed—production of bone prevails in only 62 per cent. But the arrangement of the new formed bone in radiating spicules is also much more frequently observed in the first than in the second group. In the first group it is observed in 28 per cent of all cases and in 33 per cent of the cases of this group with bone production, while in the second group it is present in 8 per cent of all cases and in 13 per cent of the cases of this group with bone production. These figures speak against the suppositions mentioned above that the frequency of the arrangement of the formed bone in spicules is in inverse

ratio to the amount of the new formed bone. It is evident that the age of the patient exerts a great influence upon the amount of new formed bone as well as on the arrangement of the bone.

In the osteolytic osteogenic sarcomata, the periosteal reaction together with the lipping of the periosteum may appear very late; therefore waiting for the typical lipping for the diagnosis is not recommended. In the usual osteolytic osteogenic sarcoma the roentgenogram shows, at first, expansion of the involved bone to a small degree. Then the bone shadow becomes spotted because of areas of bone destruction. In the print such a roentgenogram looks as if one erased the bone in spots (Fig. 48). Such cases resemble metastatic skeletal tumors with a predominance of destruction. It is in such cases that a pathological fracture is a more or less frequent complication. In rare instances of osteogenic sarcoma the destruction of the bone is more circumscribed and limited to a smaller area; this is usually observed in tumors located in or about the epiphysis (Plate 10).

A large expansion of the involved shaft is rather an exception and not the rule in osteogenic sarcoma. The old shaft is usually seen in osteogenic sarcoma passing not expanded through the fusiform tumor mass. The sclerosed shaft may even appear in the roentgenogram to be shrunken. That there are exceptions when the bone is first expanded and then destroyed has been mentioned above. Such a course is usually observed in cases with a wide

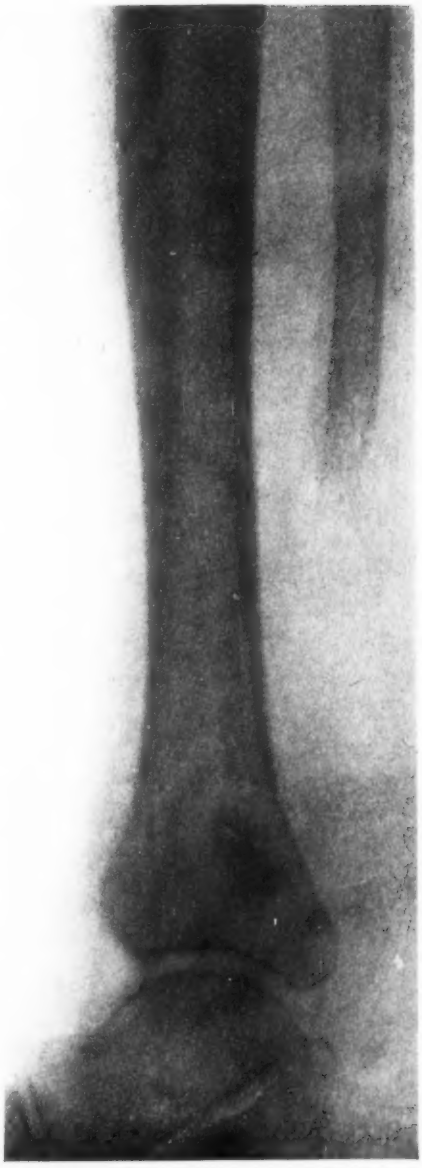


Fig. 48. Case 129. Osteogenic sarcoma in a woman 55 years old. Osteolytic variety. Death 1 year after onset.

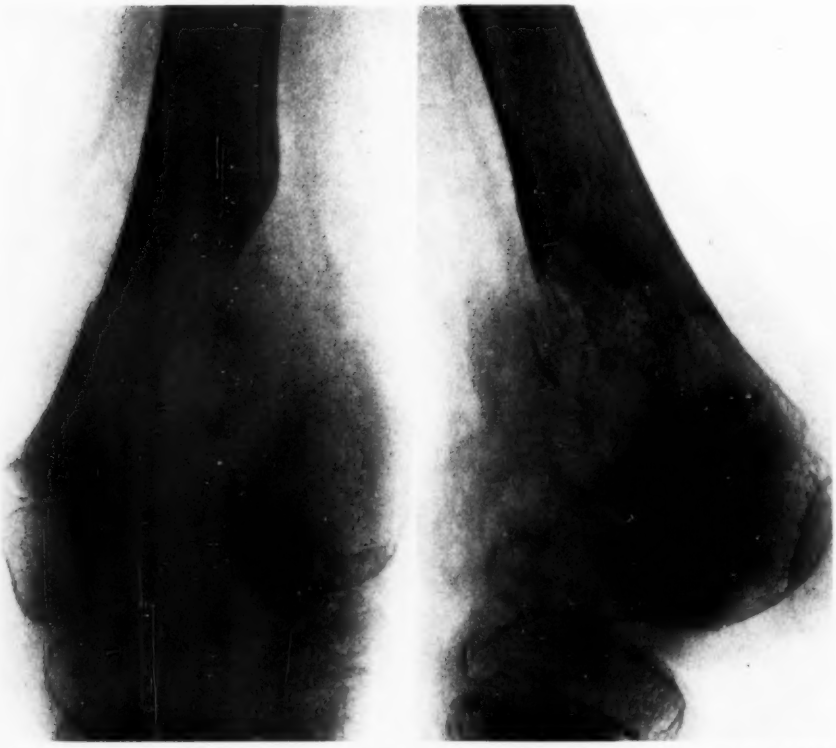


Plate 10. Case 473. Osteogenic sarcoma in a boy 11 years old. Osteolytic variety which is uncommon in the young. The tumor was curetted because of an erroneous diagnosis of giant cell tumor. Death 10 months after the onset.

involvement of the shaft of the bone, slightly resembling Ewing's sarcoma roentgenologically. The shaft of a bone surrounded by an osteogenic sarcoma and appearing normal in the roentgenogram is not to be considered free of involvement; a tumor may show up well around a bone which casts a normal appearing shadow despite an extensive involvement of the haversian system and medullary cavity. For the same reason to attempt to define the extent of involvement by roentgenological evidence alone is hazardous in surgical therapeutic considerations.

Important for the diagnosis as some roentgenological features of osteogenic sarcoma may be, it would be misleading to consider them as absolutely pathognomonic. Setting up rules in the roentgenology of bone tumors will only contribute greatly to the discredit of roentgenography as a leading diagnostic method in skeletal oncology. Most of the roentgenological diagnostic points enumerated

Fig. 49. Case 222. A benign osteogenic tumor in a man 22 years old. Onset in 1910. Local excision in 1914. The patient was well in 1923. Note the sharp outline of the base of the tumor, the uniform density of the tumor and its jagged, velvet-like dome.

above may become worthless in an attempt to differentiate from osteogenic sarcoma a skeletal lesion with the same given roentgenological feature. For these reasons it is important to review here the skeletal lesions most frequently confused roentgenologically with osteogenic sarcoma, and to stress the chief

Plate 11. Case 216. See Figure 19. Osteogenic sarcoma in a woman 27 years old. Notice the speckled appearance of the tumor in the roentgenogram. The tumor cells are highly differentiated; bone with excessive cartilage formation make up the bulk of the tumor.

differentiating points. The differentiation from osteogenic sarcoma of other primary malignant skeletal tumors as well as giant cell tumors will be dealt with in the corresponding chapters.

As was mentioned, one of the most important roentgenological features of osteogenic sarcoma is that it is rarely definitely limited. However much depends on the stage in which the osteogenic sarcoma is seen; not infrequently after intensive radiation the tumor commences to cast a quite sharply limited shadow. In these instances the roentgenogram may not be inconsistent with the appearance of a benign osteogenic tumor. However the uniform density of the benign tumor, with the dome of the tumor jagged, velvety like, because of its cartilaginous covering, will frequently help to decide the diagnosis. In a benign osteogenic tumor there will be in most cases a sharp outline of the tumor toward the bone and the surrounding soft tissues (Fig. 49). A benign osteogenic tumor has cartilage on the external surface only, in imitation of the articular ends of long bones; cartilage dispersed throughout the tumor mass speaks for osteogenic sarcoma and against benign osteogenic tumor. Lipping of the periosteum is absent in a benign osteogenic tumor. It is also very unusual for a benign tumor to present the spindle form typical of osteogenic sarcoma. There is, however, a condition which, although rare, may puzzle the diagnostician because of the spindle shaped shadow of the roentgenogram even with presence of lipping of the periosteum. It is a subperiosteal hæmorrhage in hæmophilia. The periosteum is stripped away by the extravasation, the whole process forming a fusiform swelling. The pressure of the blood upon the bone may be so high as to cause an erosion of the cortex. Such cases have been reported in the literature. Similar confusion may be caused by Moeller-Barlow's disease with the only difference that there the hæmorrhages are usually limited to the diaphyses of the long bones.

That the sun-ray arrangement of the new formed bone was present in the Registry material in only about 18 per cent of cases was mentioned above; there was also indicated that such a radiation of the new formed bone lamellæ may be seen occasionally in chronic inflammatory conditions of the skeleton. Even less dependable for the diagnosis of osteogenic sarcoma is the longitudinal striation of the new formed bone, when the old cortex seems to be frayed longitudinally. It is not unusual to find such a structure in periostitis whether traumatic or inflammatory. In traumatic periostitis, however, the outline of the shaft remains distinctly limited and the cortex uneroded by the exostoses. It is uncommon for traumatic periostitis to surround the whole periphery of the bone as does osteogenic sarcoma. That, as a result of traumatic periostitis, large tumors may appear is known from the literature. In connection with this one has to consider also myositis ossificans, which is especially confusing when accompanying an osteogenic sarcoma as it is occasionally observed in osteogenic sarcoma after sport injuries.

Plate 12. Case 533. Multiple intra-osteal chondromata in a girl 8 years old. The entire skeleton was involved. The histology was inconclusive. Heavy radiation led to local improvement.

One can be more baffled in diagnosing a skeletal lesion by the possibility of a periosteal or cortical gumma than by periostitis. The similarity between syphilitic skeletal lesions especially gummata, and osteogenic sarcomata may be so great that despite all precautions many experienced diagnosticians have met with errors at some time or other. This is sufficient reason why one should refrain in doubtful cases from attempting a diagnosis before the result of a blood Wassermann reaction or even a provocative reaction is known. There are, however, some points which may help one in differentiating between syphilitic skeletal lesions and osteogenic sarcoma. A gumma is growing less rapidly than a sarcomatous tumor; the regressive changes in gumma take place much earlier than in osteogenic sarcoma. The extensive destruction of bone by gumma in the presence of a small sized tumor and the common absence of the periosteal spindle speak well for gumma and against osteogenic sarcoma.

When the new formed bone is present as foci throughout the tumor mass the roentgenogram is characterized by a spotted appearance. A picture similar to that is observed in skeletal chondroma. The calcified foci disseminated throughout the cartilaginous tumor cause a characteristic speckled mottled appearance with the individual spots sprinkled about (Plate 11). The differentiation in the roentgenogram between skeletal chondroma and osteogenic sarcoma is feasible. The sharply outlined pedunculated base of the tumors springing from a bone without any demonstrable pathological changes and the absence of the periosteal spindle will frequently suffice to establish the correct diagnosis. More difficulties may be encountered in intra-osteal and multiple chondromata. They are frequently seen in the upper portion of the femur, about the great trochanter. In such instances the question of differentiating between these tumors and an osteolytic osteogenic sarcoma may arise (Plate 12). At variance with osteolytic osteogenic sarcoma is the thinning of the cortex in central chondromata, and also the honeycombed structure with the thin, well defined bony septa traversing in the roentgenogram the defect in the osseous tissue of the bone. Not infrequently a question arises of differentiation between an osteolytic osteogenic sarcoma and skeletal carcinomatous metastases. This is hardly possible from the roentgenogram alone, but the presence of lipping and an unexpanded shaft speak for sarcoma.

More uncommon is the necessity of differentiating between osteogenic sarcoma and a bone cyst. Such a differentiation meets with fewer difficulties. This is especially true in more advanced cases. The clinical similarity between a bone cyst and osteogenic sarcoma may be great. The rheumatoid night pains, tenderness, although less marked than in osteogenic sarcoma, and pathological fracture all may be present with a bone cyst. Thus the clinical picture of a bone cyst as it usually appears in the young greatly resembles osteogenic sarcoma and the clinical examination alone will be insufficient for an accurate diagnosis. A

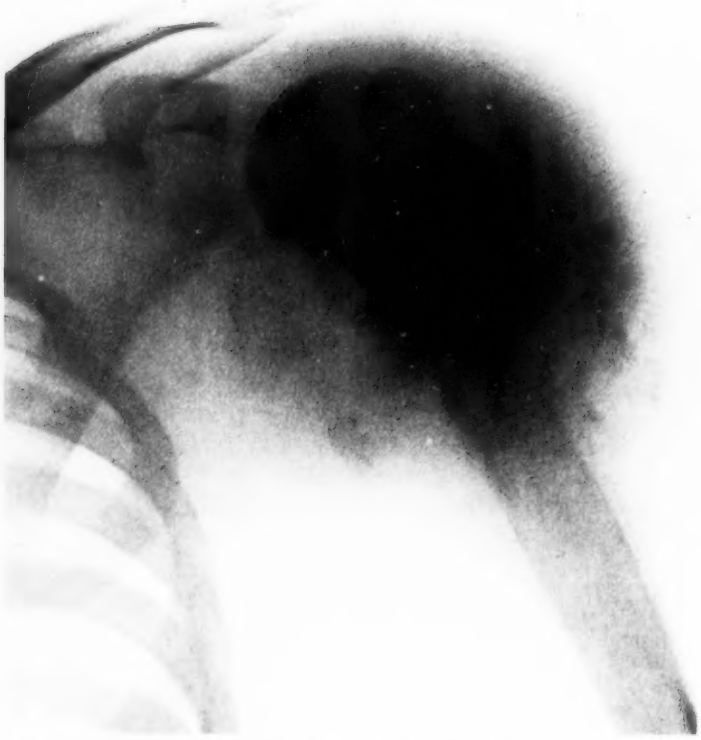


Fig. 50. Case 371. Osteogenic sarcoma in a boy 16 years old. Typical as to location and radiological appearance. The roentgenogram was taken 2 months after the onset at which time pulmonary metastases were demonstrated. Death 7 months after the onset.

roentgenogram will be almost indispensable although in the early stages a roentgenogram may be of no avail in the attempt to differentiate the lesion from an osteogenic sarcoma. The chief sign of a bone cyst is that it is always intra-osteal, i.e., central. Its presence usually leads to some expansion of the shaft; the cortex may become very thin but it is uncommon to have a perforation of the bone shell. Theoretically there is no reason why an actively enlarging bone cyst should be unable to perforate the cortex, but clinically such a perforation is a curiosity, probably because a pathological fracture interrupts the activity of the cyst when the latter is little short of a cortical perforation. When the cyst is located in the diaphysis it is usually ovoid in shape while the roentgenogram of a cyst in the ends of the bone may show a trabeculation similar to giant cell tumor.

Fig. 51. Personal observation; case not registered. Osteogenic sarcoma in a girl 8 years old. Death 9 months after the onset. The shaft of the bone is seen passing through the tumor which surrounds it. The periosteal spindle and the periosteal lipping are seen. The tumor stops abruptly at the epiphyseal junction.

In the roentgenogram, the condition most frequently confusing the diagnosis of a malignant bone tumor is chronic low grade osteomyelitis. Because it is most frequently diagnosed erroneously in the presence of Ewing's sarcoma, the points differentiating it from malignant skeletal tumors will be discussed in detail later. The differentiation from skeletal tuberculosis will rarely cause difficulties in the diagnosis. The fact that osteogenic sarcoma is generally met with in young adults in apparently the best of health is in great contrast to tuberculosis which is usually observed in persons with a general condition below par. Sarcoma and tuberculosis also differ in location; e. g. osteogenic sarcoma very rarely if ever begins in the epiphysis of the bone while tuberculosis nearly always involves the joint end of the bone and primary diaphyseal tuberculosis, although known, is exceptionally rare. While in sarcoma the overlying skin is reddened early because of dilatation of superficial veins, in tuberculosis the skin is usually paler than normal.

Roentgenograms of osteogenic sarcoma in the same location in patients of about the same age reveal certain common characteristic features which may be best appreciated from a study of typical instances. I have attempted to present here as illustrations roentgenograms of characteristic types of osteogenic sarcoma

Fig. 52. Case 269. Osteogenic sarcoma of the myxomatous variety in a man 68 years old. Showing slight expansion of bone and a trabeculation of the medullary cavity.

Fig. 53. Case 535. Osteogenic sarcoma in a man 28 years old. The speckling in the roentgenogram is typical of osteogenic sarcoma of the upper femur, in the trochanteric region, due to excess of cartilage and calcification foci in it. Death 11 months after onset.

(Figs. 50, 51, 52, 53; Plate 13). Especial mention should be made of the variety of osteogenic sarcoma which is situated in the diaphysis of the femur. As it was pointed out above, about 8 per cent of all osteogenic sarcomata of the femur have such a location.

Plate 13. Personal observation; not registered. Osteogenic sarcoma in a boy 18 years old. Roentgenogram (left) taken 3 months after onset. An exploratory operation was performed at that time and the pathologist reported "osteomyelitis." Right, Roentgenogram taken 7 months after the onset.

It is with great care that a diagnosis of osteogenic sarcoma should be made when a tumor involving the midshaft rather than the end of the femur is met in a young individual, since such a location is characteristic of Ewing's sarcoma. A differentiation of such an osteogenic sarcoma from Ewing's sarcoma can hardly be made from the roentgenogram, save in the far advanced stage; a microscopic study is indispensable here. A careful follow up of the clinical course of this variety of osteogenic sarcoma seems to indicate that the tumor passes through two phases. The first phase is the osteoblastic; the bone is slightly expanded but distinctly denser than normal and no distinct tumefaction can be made out as yet. Then the disease enters the second phase; the bone becomes considerably widened, and at one point or another a rapid destruction of the bone commences (Plate 14). The widening of the shaft may be accompanied by the appearance of longitudinal striations (Fig. 54). With the progress of bone destruction a large extra-osteal tumor appears. The bone destruction is so rapid that a pathological fracture is not uncommon in these cases; the frequency of spontaneous fractures probably being augmented by the situation of the tumor in the middle of a long, weight-bearing bone. Occasionally myositis ossificans may be confused with this variety of osteogenic sarcoma. Unlike osteogenic sarcoma, myositis ossificans usually develops during the first three or four weeks after the onset with trauma with a subsequent period of quiescense. The consistency of the tumor in myositis ossificans is bony hard in contrast to the firm but resilient feel of osteogenic sarcoma (Plate 15).

While an attempt to diagnose a skeletal lesion suspected of malignancy from the clinical and roentgenologic angle does not involve any procedures which are apt to influence the natural course of the tumor in any way, an examination from the pathological angle is possible only after direct encroaching upon the tumor mass. This fact alone would seem to narrow the clinical importance of the pathological angle of examination as a preliminary step toward diagnosing the skeletal condition. The uncertainty of a clinical examination and the diagnostic limitations of roentgenology, however, foster the importance of the pathological angle of examination in these tumors. The frequently encountered regressive changes, spontaneous or after radiation, inflammatory changes, traumatic or of infectious nature, and various peculiarities of the histology of osteogenic sarcoma make it desirable to diagnose a malignant skeletal tumor from the gross appearance, without dependence upon the microscopical picture. As it is easy to see from the gross anatomy of osteogenic sarcoma, the prerequisite of such a diagnosis is wide experience in this relatively rare disease. A pathological diagnosis of malignant skeletal tumors is not always dependable and the personal equation of the pathologist counts greatly in the diagnosis; sometimes even a diagnosis based on examination of numerous slides of a bone tumor may not be correct. Such examinations are notoriously unreliable when the material to be examined

Plate 14. Case 639. Osteogenic sarcoma in a girl of 12. Notice location in the midshaft of the femur. Roentgenograms taken one month after onset showing bone denser than normal; and 4 months after onset showing tumefaction and extensive destruction of bone.

Fig. 54. Case 638. Osteogenic sarcoma in the midshaft of the femur in a girl 14 years old. Showing the combination of features of osteogenic sarcoma—radiating spicules; and of Ewing's sarcoma—the widening of the shaft and parallel striations. Death 3 years after onset.

Plate 15. Case 263. Myositis ossificans in a man 34 years old. The onset in July, 1919, with a tumor of the fibula (roentgenogram 1). In November, 1919, the involved part of the fibula was resected, but in February, 1921, a recurrence was noticed (roentgenogram 2). In February, 1923, an amputation was performed (roentgenogram 3). Notice the striations of the ossified muscle bundles. This case was interpreted by some pathologists as an osteogenic sarcoma.

consists of tumor tissue removed in biopsies. I have seen cases of osteogenic sarcoma in the Registry material in which repeated biopsies, even five in the same case (616), were made to secure tissue for diagnosis while the correct diagnosis became obvious after all these explorations only from the rapid growth of the tumor infected during the exploratory operations. It is easy to understand why, when the differential diagnosis of bone tumors is uncertain by any single method, there exists a tendency to "explore" the tumor and to shift the task of diagnosis to the pathologist. Although a biopsy of a bone tumor is frequently advocated by surgeons, it is significant that pathologists most experienced in bone tumors raise their voices against probatory incisions in cases of suspected malignant bone tumor. It would seem that the attempt to neutralize the bad effects of a probatory incision by intensive cauterization of the operative wound is unavailing, since the dangers of an exploratory incision are not merely the possibilities of dislodgement of metastases but also the surgical insult combined with the irritation of the escharotic used which may increase the rate of growth of the tumor. Cases have been seen in which an infection brought in during such an exploratory incision led to a stimulation of the growth of the tumor. It is true, of course, that frequently an exploration will relieve the patient of pain because of decrease of the tension beneath the periosteum, following the evacuation of sarcomatous tissue; but soon the tumor will begin to grow rapidly. If an exploratory operation is decided upon, it should be remembered that in tumors previously irradiated it may be difficult to secure a sufficiently vital and representative piece of tumor to warrant a histological diagnosis. In removal of tissue for diagnosis all the removed tissue should be saved, because the recurrent tufts mixed with granulation tissue and infection are dangerous for a histological examination and may lead to an erroneous diagnosis and sacrifice of the patient's limb in the presence of a benign tumor.

As a substitute for the harmful exploratory incisions in doubtful cases of bone tumor a therapeutic radiation test has been suggested. Recent experience has furnished sufficient evidence to justify the belief that radium or roentgen-ray radiation may frequently replace a biopsy. It may be safely stated that no skeletal lesion responds so rapidly to radiation as do certain types of malignant tumors. The most agonizing constant pain frequently disappears after one or two exposures. The rate of growth slows down remarkably so that a speckled and hazy roentgen-ray shadow of a diffusely and rapidly growing osteogenic sarcoma soon acquires a more or less well limited outline; the shadow becomes denser, more compact (Plate 16). The most rapid response to radiation is noticed in Ewing's sarcoma and cellular osteogenic sarcoma. The response is less frequent and prompt in acellular and highly ossifying tumors, although the subjective relief is frequent also in this variety. It is fully ascertained that a preliminary radiation test will frequently help one to arrive at a diagnosis without an exploratory incision. For

Plate 16. Personal observation; not registered. Osteogenic sarcoma in a boy 5 years old. Death 9 months after the onset. Showing the response of the tumor to radiation. Between the two roentgenograms an interval of 3 weeks has passed, during which time the patient was given 3 half hour exposures to 200,000 volts of roentgen rays.

better evaluation of the results of a radiation test it is important to have the roentgenogram of the lesion before and after radiation of equal density. To secure the latter it should be remembered that the density of a roentgenogram is the result of the intensity of exposure which is directly proportionate to the current, to the square of voltage, and to the time of exposure and indirectly proportionate

to the square of the distance of the tube from the film. The density also depends upon whether screens are used or plain films.

Therapy

In bone sarcoma as in other malignant tumors the question of the therapy is still awaiting its answer. It is a strange fact that with our knowledge of minute details of the histopathology of bone tumors the progress along the practical therapeutic road is almost in the same stage that it was in some fifty years ago. As a rule malignant bone tumors are fatal and we know of no therapeutic method to prevent death from this disease. Occasionally one sees in the literature the term "cure" in connection with bone sarcoma. The word "cure" is not to be taken in the real sense of the word, usually a five years' cure is understood; this term is valuable only in comparative studies. Patients with osteogenic sarcoma who were free of metastases for years after amputation have later died of pulmonary metastases. It is an exaggeration to say that a tumor from which the patient has recovered is not a malignant bone tumor; but in such a statement there probably is more truth than error. The relative individual character of the term "cure" is one of the reasons why all statistical data of cure from bone sarcoma through one or another method of treatment are nearly worthless. Too many subjective, unstandardized factors are in play in such statistics to render them of value; apart from what is meant by "cure" much depends upon the stage of the disease when treated, upon the method and character of treatment and upon other changeable factors. Mistaken diagnoses are not infrequent in such reports. Cases of osteitis fibrosa, skeletal syphilis, and other bony lesions, erroneously interpreted as sarcoma will obviously raise the frequency of "cure." Of the same value are also the reports of spontaneous cure of bone sarcoma. It is true that authoritative observers have described regression in growth and development after partial surgical removal but no authentic cases of malignant bone tumor can be found among the reports of spontaneous cures.

To the end of the past and the beginning of the present century belongs the origin of the immuno-therapeutic attempts to cure or relieve patients with malignant tumors including bone sarcoma. The sponsors of these therapeutic measures set out from a viewpoint that malignant tumors are of a parasitic infectious nature. These attempts are also based upon observations of spontaneous disappearance of malignant tumors after an erysipelatous infection of the patient. The toxin and sera therapy of malignant tumors form a most romantic chapter in the history of these diseases. Being enthusiasts, the authors were easily impressed by doubtful results readily explainable on other grounds. And truly, what could be more enchanting than to make the organism immune to the infectious, fatal tumor? One after another the various, ingenious methods of immunization and detoxication were raised merely to fall discredited.

From all the various attempts at specific therapy we have today left only Coley's mixed toxins. The theoretical idea underlying Coley's toxins is active immunization of the patient. After long experiments with numerous preparations of various streptococci with and without the combination with bacillus prodigiosus, Coley arrived at the following way of preparing his toxins. A ten day old agar culture of bacillus prodigiosus is sterilized by heat and added to a sterile three weeks old streptococcus broth culture in the approximate proportion of 100 cubic centimeters of streptococcus culture, 30 cubic centimeters of suspension of the prodigiosus culture, and 20 cubic centimeters of glycerin. This preparation is introduced subcutaneously or intratumorally. It is Coley's belief that if, after four weeks of intensive treatment with the toxins, no marked improvement is registered, then the expectations from this therapy are poor. When the tumor responds to the treatment, Coley recommends continuing this therapy for a year or longer. The usual reaction to a foreign protein, fever, chills, dyspnœa, vomiting, and general malaise, are not infrequently observed. From a study of the abundant literature of this subject and of the material of the Registry one cannot agree with the enthusiastic reports of Coley. Many advanced and inoperable cases which have responded well to Coley's treatment have proven to be giant cell tumors for which it is not very unusual to see spontaneous improvement after a pathological fracture. However, whatever one may think of Coley's toxins one is amazed at the sincere honesty and tireless ambition with which Coley has worked on the development of this method of treatment.

Of late we frequently hear of the importance of Coley's toxins in combination with roentgen or radium radiation. The fact that this new wave of enthusiasm for the toxin takes place in days when radiation is brought into extensive practice is apt to make very dubious the importance of the toxins in themselves. There is no proof, however, to deny the validity of the argument that toxins increase the effect of radiation. The question of the way Coley's toxins could influence the tumor is too obscure to be discussed here. A general reaction of the organism to a foreign protein is probably the best explanation. During the long history of the therapy of bone sarcoma there was a time when chemical therapeutic methods were in vogue. It is safe to say that nothing has been achieved in bone tumors from chemotherapy; formalin in 10 per cent solution is occasionally used in inoperable ulcerated tumors as a deodorant.

Until recently operative therapy of malignant bone tumors has been the method of choice. This despite the fact that with the highly developed surgical technique and skill it is still impossible by operative treatment to influence the metastases of the tumor latent in other organs. It is hardly necessary to argue here the evident fact that radical surgery alone is unable to solve the therapeutic question of bone sarcoma. At first glance it is strange that, although the experience of radical surgery has been disappointing, it has until recently dominated all

therapeutic considerations in bone sarcoma. In their attitude toward the question of operative treatment of bone sarcoma two extreme types may be observed among surgeons. To one belong those who, at a slightest suspicion of bone sarcoma, amputate the extremity. Their argument is plain; it is better to sacrifice a limb than the whole organism. Frequently in these cases the surgeon never knows the true nature of the lesion; all goes well, and the number of "cures" from surgical treatment is large. To the other extreme belong surgeons who will doubt the diagnosis of malignancy in the absence of any of the characteristic symptoms of the disease, frequently despite a histological diagnosis. To these surgeons malignant bone tumor also spells amputation, a most radical operation to avoid which the diagnosis is doubted until, as a court of last resort, amputation is performed. Procrastination and delay here make the amputation entirely futile from the start.

From an analogy between bone sarcoma and carcinomatous tumors a question suggests itself: could not an early diagnosis combined with radical surgical treatment solve the problem of therapy in bone sarcoma as it occasionally does in some types of early diagnosed and well localized carcinomatous lesions? There is abundant evidence on hand to the effect that nothing is known that would help to establish a diagnosis of bone sarcoma in an incipient stage. Instances of osteogenic sarcoma are occasionally seen in which in spite of the fact that an amputation is made a few weeks after the subjective onset of the disease, death follows from pulmonary metastases. This would seem to indicate that a metastatic dislodgment of a malignant bone tumor is incomparably more rapid than in malignant tumors in soft organs or, what is more probable, that the subjective onset does not correspond to the true onset of the disease, which passes on unnoticed and gives rise to metastases before it attracts the attention of the patient.

In the consideration of the question of the indications for radical surgical treatment in a case of osteogenic sarcoma there are certain conditions which play a decisive rôle. The main condition is certainty of the diagnosis. In the large majority of cases the experienced diagnostician will arrive at a definite diagnosis from a careful analysis of the clinical and radiological data and of the result of the therapeutic radiation test. There will be, however, in exceptional cases times when the experienced observer will be unable to arrive at a definite diagnosis; in these cases an exploratory incision will be unavoidable. In such cases the surgeon must be prepared to act according to the diagnosis, and before operation he must have the consent of the patient to amputation. To explore simply for diagnosis and then to institute conservative treatment in case of malignancy is one of the most radical mistakes made. For the clinician well acquainted with the gross pathology of bone tumors, an exploration with an application of two constrictors is of real value; the distal constrictor is to prevent dislodgment of

Plate 17. Courtesy of Dr. Baker, Newark; not registered. Osteogenic sarcoma in a boy 16 years old. The roentgenogram was taken 2 weeks after the clinical onset of the disease. Very little is to be seen in the roentgenogram except for a slight jagging of the internal aspect of the femur (arrow). A roentgenogram of the chest made on the same day revealed far advanced pulmonary metastases.

metastases from the area of exploration while the proximal constrictor is for an immediate amputation should the tumor be found malignant.

The second condition in the consideration of indications for radical surgical treatment is the old rule in surgery of malignant tumors, "all or nothing." Do not operate if you think that complete removal and eradication of the primary tumor is impossible. This rule alone excludes surgery in osteogenic sarcoma of the spine, skull, and pelvis.

Another condition upon which an indication of surgical treatment is based is absence of clinical evidence of metastatic generalization of the tumor. A radiological examination of the lungs is required before instituting surgical treatment in osteogenic sarcoma (Plate 17); small metastatic foci which cannot as yet be perceived by percussion and auscultation will frequently be demonstrated in the radiogram as light, cloudy spotting in the normally dark lung shadow in the film. A preliminary radiological examination of the whole skeleton will occasionally save the surgeon from disappointment when multiple tumors or bone metastases are present. As exceptions to all these requirements for indication of radical surgical treatment are patients suffering unbearable pain which cannot be ameliorated by radiation. In such patients surgical treatment should be considered even in the presence of features which in an ordinary case will contraindicate such treatment.

After the indication for operative treatment has been established a question will arise of choosing between a conservative surgical procedure and a radical one. This question is almost as old as surgical therapy of malignant bone tumors itself. It was Mikulicz who first emphasized the advantages of tumor excision and bone resection over amputation and disarticulation. There is little if any difference between these two methods of surgical treatment, radical and conservative, as far as metastases are concerned, and if the resection or excision of the tumor is well done there is no reason why metastases should be more frequent than after an amputation. However, the main motive pointed out by Mikulicz, that a patient will sooner meet the necessity of a resection than a crippling amputation has lost ground at the present time with the high development of prosthetic surgery; it is indeed well known how very frequently an artificial limb is of incomparably more use and comfort to the patient than a leg with a stiff joint or with a constantly aching bone. Bloodgood has frequently mentioned that it is not unusual for his patients to become more efficient after an amputation; an artificial limb is frequently well borne by young patients even in dancing. It is true that in many cases the tumor can be removed just as completely by resection as by amputation; however, the hazards of the operation in connection with the dangers of overlooking involved tissue are great enough to strip the method of resection of all advantages over a more radical procedure, and unless the tumor involves a region for which no satisfactory artificial limb can be fitted, amputation is to be

preferred to any conservative surgical procedure. To resect the tumor and graft bone in a patient with an osteogenic sarcoma is an unwise procedure, not in accord with the palliative character of the operative measure.

If the choice between resection and amputation can be argued by a surgeon experienced in bone pathology, it is entirely out of place when the patient is in the hands of the surgeon operating only occasionally for bone tumor, for whom the method of choice is amputation or disarticulation if operative treatment has been decided upon. But even before the experienced observer decides to excise or amputate, it is advisable as a preliminary step in the operation to explore the main blood vessels, since if these are surrounded by tumor, amputation is the only choice. It is significant that the surgeons most experienced in bone tumors at the present almost wholly prefer amputation to resection or excision.

Excision usually comes up in borderline cases of bone tumors like skeletal chondroma. With this tumor any partial, conservative operative procedure is dangerous; indeed, no operation which leaves a trace of these quasi pedunculated tumors should be advised. It should be remembered that the apparent gross integrity of the cortex of the bone to which the tumor is attached is not dependable. In excising these tumors it is best to chisel away liberally the cortex upon which the base of the tumor rests.

Another conservative surgical procedure which dates back at least 50 years is ligation of the main artery supplying the region of the tumor involvement (Plate 18). In the literature one finds indications that sometimes such a ligation in a telangiectatic sarcoma leads to healing of the tumor for years. Recent attempts to influence a very vascular sarcoma by means of ligation of the afferent artery were unsuccessful, thus throwing a shadow of doubt upon the true sarcomatous nature of the tumors reported in the old literature.

Whatever the operative procedure decided upon, whether amputation or excision, it should be kept in mind that in muscles the spreading of sarcomatous tumors may easily be overlooked, and since the tumor, after perforation through the investing capsule, rapidly spreads along the intermuscular septa an extensive dissection of the muscles is most important. If operation is going to help temporarily, amputation of the involved bone may be sufficient, and a disarticulation of the proximal joint is unnecessary. Death takes place not from a recurrence because of a low seat of amputation but because of lung metastases which cannot be prevented by disarticulation. I have seen cases in which midthigh amputation in osteogenic sarcoma of the lower end of the femur was not followed by recurrence locally but by pulmonary metastases. Occasionally one is, however, misled by the roentgenogram if one depends upon it entirely in choosing the seat of amputation; since the medullary cavity may be stuffed with tumor while in the roentgenogram this portion of the bone will appear normal (Fig. 55). Here again one sees the necessity for the surgeon undertaking surgical treatment of

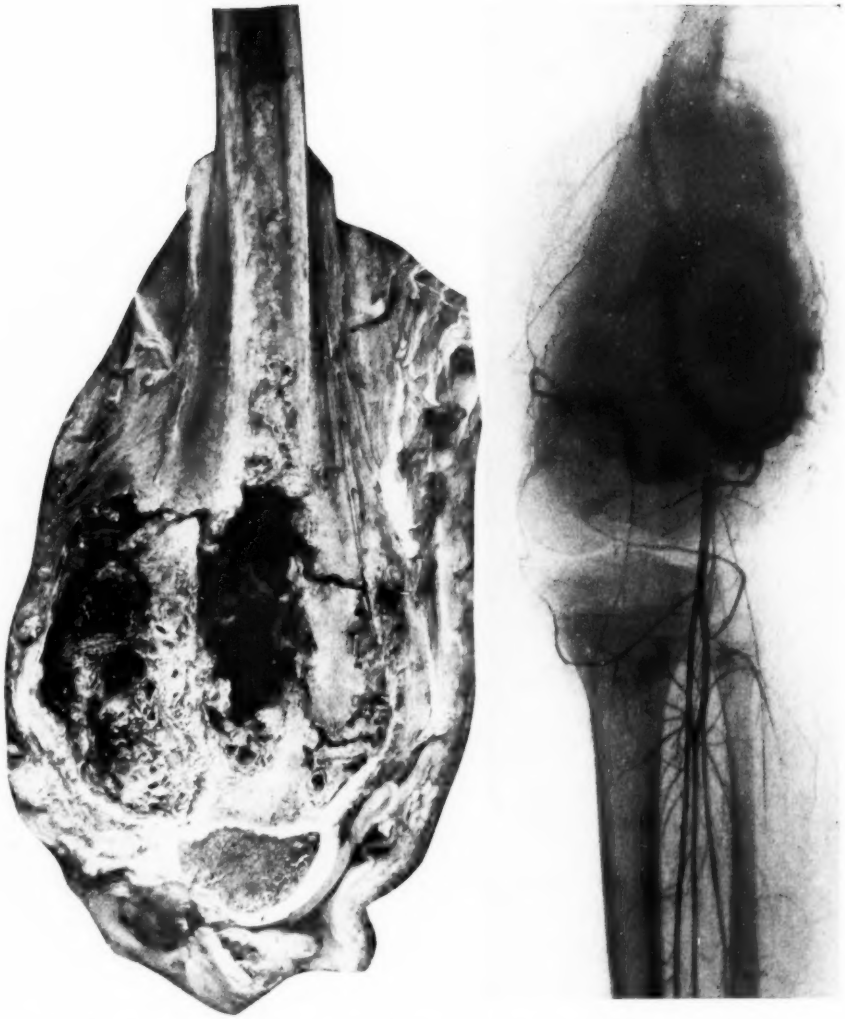


Plate 18. Case 508. Same case as that in Figure 7. Osteogenic sarcoma in a girl 5 years old. A very vascular "telangiectatic" tumor in which ligation of the afferent artery as a therapeutic measure suggests itself.

bone tumors to be well acquainted with the gross anatomy of these lesions. The ease with which an involvement of bone-marrow at the point of amputation may be overlooked explains the preference for disarticulation over amputation among surgeons years ago. Experience has shown, however, that the results

Fig. 55. Case 382. Osteogenic sarcoma in a boy 18 years old. Roentgenogram was taken 4 months after the onset; it shows an intact outline of the cortex of the bone. Disarticulation was performed at the same time and an extensive sarcomatous involvement of the medullary cavity was found; the involvement extended down past the middle of the shaft. Death 7 months after onset.

after disarticulation of the hip do not differ from amputation distal to the hip joint as far as secondary growths and recurrences are concerned. On the other hand, in exceptional instances even disarticulation is not sufficient for avoiding a local recurrence; for example, in sarcomata of the upper half of the humerus where a complete shoulder girdle extirpation (amputatio interscapulo-thoracalis) promises far better results as far as local eradication of the tumor is concerned.

The vast accumulation in recent time of technical knowledge and the advance in the application of radiation, roentgen ray and radium, in the therapy of malignant tumors in general required a re-evaluation of all accepted principles and traditions as far as the therapy of bone sarcoma is concerned. Even now clinicians are inclined to think that if a bone sarcoma is operable there is an indication for surgical treatment without losing time in radiation, while for radiation treatment only the inoperable cases are to be left. That this view is obsolete and not in accord with the disappointing results in radical surgical treatment is obvious. It is true that no astounding success has as yet been achieved by radiation therapy of malignant bone tumors and that most results here are unsatisfactory; however, the evidence on hand is encouraging, and radiation is as legitimate a therapeutic procedure in bone sarcoma as radical surgery.

Radiation therapy is still in the experimental stage, and institutions lacking the best available modern apparatus and specially qualified personnel should refrain from making wide use of it. Unfortunately we are witnessing at the present time a wide popularity of radiation therapy among persons lacking the necessary experience and skill. It is obvious that since, to the average inexperienced radiologist, the possession of an X-ray machine and radium means he is qualified to administer radiation, considerable abuse and misuse of radiation in bone tumors may be expected. Only with a vast clinical material and under prolonged, careful observation will this promising method of therapy become definite. Abuse of radiation by incompetent persons will not only greatly contribute to the confusion already overwhelming this new field of medicine, but it is also connected with great dangers for the patient. That errors in technical application of radiation may do harm is a fact fully ascertained. Whatever the true biological cause of the influence of radiation upon living tissue may be, it is attested that this action will be stimulation or inhibition depending upon the dosage together with technical factors from a detailed discussion of which I must refrain here. That inefficient radiation may stimulate the tumor growth has been emphasized repeatedly. On the other hand, over-radiation is dangerous for the normal structures of the organism. Excess of radiation may lead to ankylosis of an exposed joint. The skin becomes bronzed and leathery, and ulceration of the skin may ensue. The bones become sclerotic, ivory-like, and spontaneous fractures of such bones evince a definite delay in union. The marrow cavity becomes obliterated from over-radiation (Plate 19). Obliteration of capillaries

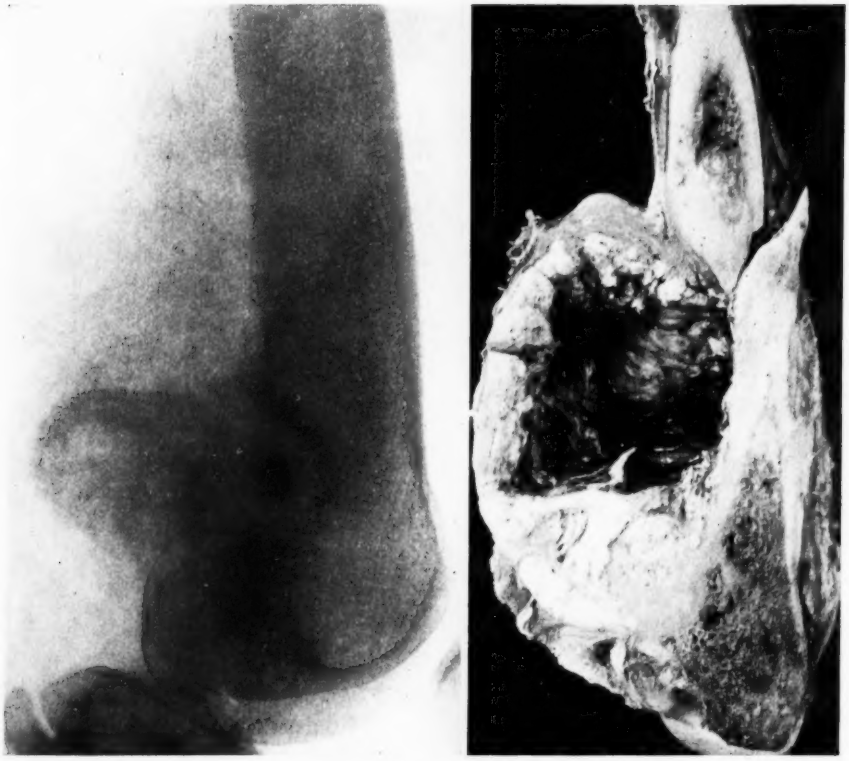


Plate 19. Case 365. Osteogenic sarcoma in a woman 25 years old. Excessive roentgen-ray radiation rendered the bone sclerotic and brittle with a pathological fracture as a result. The medullary cavity of the bone is narrowed; extensive degenerative changes in the tumor are seen, probably from radiation.

of the periosteum and endosteum causes a scarcity of callus formation in fractures. All these changes following over-radiation may be accompanied by severe pain. It is hardly necessary to argue here that in such complex biological processes as the influence of radiation upon the tumor and the organism no hard and fast rules about the dosage can exist. The reaction to radiation of similar tumors in two individuals may be very different. Only the experienced radiologist is qualified to decide the question of the dosage in each individual case. Among the various methods of application of radium the intra-tumoral application of radium recently has gained a recognized place. This procedure is hardly justified; it carries the dangers of an exploratory incision without its advantages.

The influence of radiation upon the general condition of the patient was mentioned elsewhere, as well as the influence upon the tumor itself. Radiation

leads to a conversion of a cellular tumor into a quiescent bony mass, which in large vascular tumors may contain hæmorrhagic cysts and necrotic material. In tumors with an abundant intercellular substance the response to radiation according to the present-day technique is not as effective, however, instances of calcification and ossification of the tumor combined with a retardation of growth of the tumor from radiation have been reported and are very encouraging. It is true that the cessation of growth of the tumor from radiation is merely temporary and the patient dies from local recurrence and pulmonary metastases. Thus radiation is as disappointing as radical surgery, but the difference in the sacrifice the patient has to make for radiation as compared with radical surgical treatment is enough to justify the former procedure especially since it is still in the experimental stage, when the last word has not as yet been spoken. When we learn more about the biology of radiation and acquire the same skill in administering radial therapy as we possess in using the scalpel we may hope for far better results from radiation.

The influence of radiation upon metastases is worthy of notice. I have seen cases in which the radiological evidence of pulmonary metastases has entirely disappeared from radiation and not returned for over two years; more frequently one sees pulmonary metastases kept checked by repeated radiation of the chest (Plate 20). A direct prophylactic radiation of the lungs is always indicated without regard to the method of treatment of the primary tumor whether by surgery or radiation.

Along with the rapid response to radiation of cellular and vascular osteogenic sarcomata there are some varieties which are little affected by radiation or not affected at all. To these belong tumors with a preponderance of myxomatous tissue, the skeletal chondromata and the so-called sclerosing osteogenic sarcomata. In other words, only if the tumor did not proceed in its differentiation farther than the myxomatous stage it is influenced by radiation. Moderate but persistent radiation may occasionally cause further calcification of a skeletal chondroma; radiation here leads to shrinkage of the blood supply while repeated partial surgical operations tend to increase the blood supply of the tumor. However, experience shows that skeletal chondroma of the recurring type is not suitable for radiation and should be attacked radically by surgery.

It is easy to see from the above that no generally accepted routine solution of the problem of treatment of osteogenic sarcoma is possible at the present time; and while some authoritative observers advocate amputation followed by prophylactic radiation of the lungs, others treat the patient with radiation and keep him comfortable; they think that the general comfort and well being of the patient while he is living is greater under radiation than if amputation is done. From the observations made in the foremost institutions of the country during recent years one is strongly impressed by the need of intelligent co-operation

Plate 20. Case 523. Osteogenic sarcoma. Illustrating a disappearance of pulmonary metastases after radiation. The primary tumor was in the left iliac bone in a woman 35 years old; the onset in January, 1920. In January, 1921, an exploratory incision was made. The diagnosis of osteogenic sarcoma was proven by the extensive pulmonary metastases which were demonstrated clinically and radiologically in December, 1922. Heavy roentgen radiation was followed by a marked constitutional reaction lasting 3 weeks, after which time the lungs began to clear up. Roentgenograms taken December 26, 1922, (left) and January 17, 1923.

between the clinician, radiologist and pathologist in combating this grave disease. Without desiring to seem dogmatic I find the following way probably best in caring for a patient with a typical non-sclerosing osteogenic sarcoma. Immobilization of the extremity and recumbency are followed after a few months of efficient radiation by amputation. Moderate prophylactic radiation of the chest is to be continued throughout the whole course of the disease. It is important to avoid over-radiation. Borderline cases should be treated as typical osteogenic sarcoma. Occasionally in far advanced cases immediate amputation may give the patient a few months respite from invalidism and agonizing pain.

Prognosis

A prognosis as to life expectancy in bone sarcoma is only of relative value since the natural history of the tumors which come under observation is usually changed by surgical and other therapeutic procedures. Prognosis based solely upon results of surgical treatment is of questionable importance because many individual factors, like the type of tumor, the stage of development, individual judgment in selecting cases for surgical treatment, all have a close bearing on the final result. In general no blanket rules can be made as to the prognosis of bone sarcoma. The attempts of pathologists to form a prognosis of a tumor from its pathological and morphological features alone has nowhere failed more than in bone tumors. Now and again one sees two cases with a very similar histology, one quickly ending fatally and the other going on for a much longer period of time. The histological structure, to be sure, is important since it indicates the potential malignancy of the tumor but this is merely one link in the whole chain of circumstances which render a tumor malignant. In this chain much depends upon pure clinical evidence. Of prime importance also are the gross pathological features of the tumor; they are easier to interpret, less fallacious and therefore more reliable than the histological structure, especially in the hands of those who have had a limited experience in the histopathology of bone tumors. When in malignant bone tumors the clinical course is not in accord with the histological characteristics, this discrepancy is frequently a result of deficient study of the clinical data and the gross appearance. To be of value prognosis should be determined in the light of all data available; the clinical, radiological, and pathological. The most important points of the clinical, radiological, and pathological examination for prognosis have been emphasized in the corresponding chapters; only a few points requiring greater stress will be mentioned here.

The age of the patient with an osteogenic sarcoma is important in determining the prognosis, since adolescents are less able to withstand the disease than persons above 30 years of age. In children the prognosis is still worse because of the rapidity of growth and the early appearance of metastases. The grave prognosis in the young is well in accord with the repair-and-loss-of-growth-

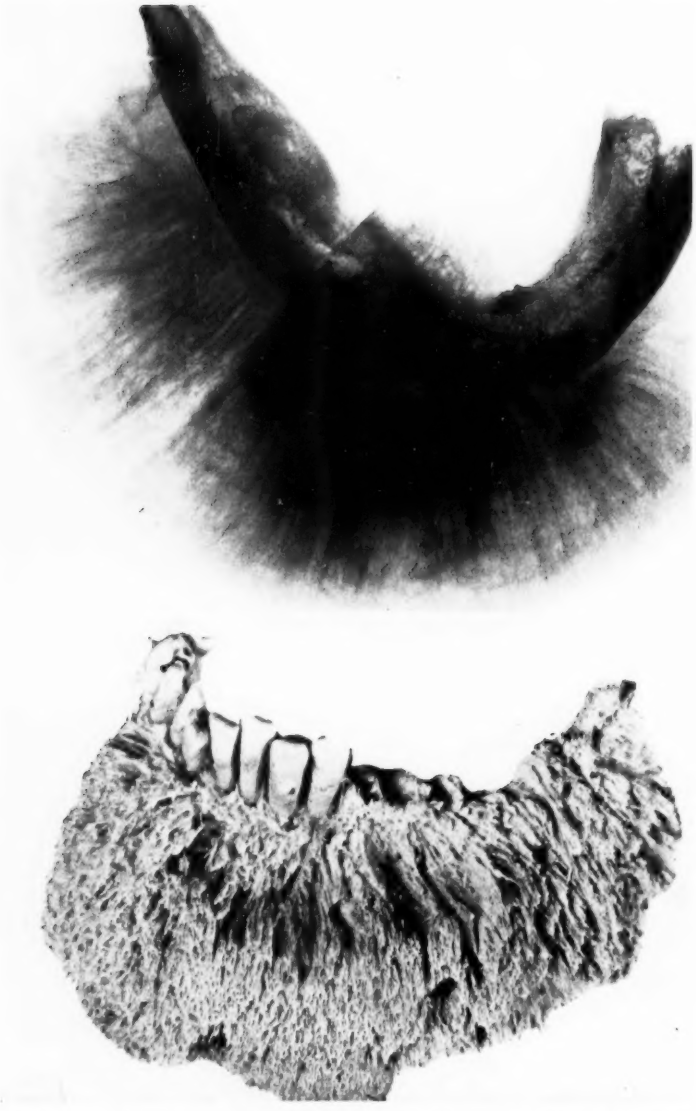


Plate 21. Courtesy of Warren Museum, Boston. Osteogenic sarcoma of the mandible. Classical roentgen-ray appearance. The photograph of the macerated and dried specimen shows the ray-like lamellæ of bone forming the skeleton of the tumor.

Plate 22. Case 404. Osteogenic sarcoma in a man 25 years old. Typical specimen and roentgenogram.

restraint theory of the origin of osteogenic sarcoma; if the tumor is a result of repair in the presence of a loss of growth restraint, then in the young a similar repair is far more dangerous than in the adult. The next features of prognostic importance are rapidity of growth and duration of the disease. A tumor of long standing is usually slow growing and is apt to be benign; however, the rate of growth may be entirely altered by trauma, accidental or surgical, after incomplete removal or exploratory incision. Extremely malignant osteogenic sarcomata usually are rapidly growing from the start. All other conditions being

equal, prognosis will change with the location of the tumor. The statistics frequently produced in the literature demonstrating the malignancy of a tumor in relation to its location are too dogmatic to be of real prognostic value. It is, however, sufficiently ascertained that in osteogenic sarcoma situated in or near the trunk the prognosis is very grave. Of extremities the gravest prognosis have sarcomata situated in the femoral neck. This is partly due to the thickness of the soft tissues covering the bone and consequently the late appearance of objective signs. Osteogenic sarcoma here if operated on has a worse prognosis than when situated in the upper portion of the humerus; the technical possibility of extensive removal of soft tissues infiltrated by extensions from the primary tumor is much greater in tumors of the upper end of the humerus than in the corresponding portion of the femur. The advisability of substituting the simple disarticulation of the humerus, in involvement of its upper end, by a complete removal of the shoulder girdle was mentioned elsewhere. The same operative procedure does not appreciably influence the prognosis in osteogenic sarcoma of the scapula. The indication in the recent continental literature of five years' cure in about 30 per cent of cases of "bone sarcoma" of the scapula are misleading since the authors seemingly did not discriminate between the types of tumors included in their reports. As a matter of fact, no osteogenic sarcoma of the scapula in the Registry has lived over three years after a radical removal. Of equally serious prognosis are osteogenic sarcomata of the clavicle, despite the fact that the clavicle can be easily removed without greatly impairing the function of the extremity. Sarcoma of the bones of the feet and hands are not so aggressive; they are early accessible for surgery and especially for radiation. The better prognosis here is perhaps due to the fact that a true osteogenic sarcoma of these bones is rare, most tumors here being extraperiosteal or of the surrounding soft tissue, with a fair prognosis. The osteocartilaginous tumors of the jaw are supposedly less malignant than similar tumors of long bones. This is probably due to the slower growth of the tumor in the jaw. Here recurrences after surgical removal are more frequent than distant metastases. The difficulty of removing the tumor which in the jaw is rarely well limited in a way that would insure against a local recurrence is probably a satisfactory explanation of it; recurrences are observed before distant metastases are recognized (Plates 21 and 22).

Not infrequently one is forced to determine the prognosis after the patient, operated on elsewhere, presents a recurrence or metastases. In itself a recurrence is no absolute proof that the original tumor was malignant. Incomplete removal of a benign tumor may result in a recurrence. That malignant tumors recur more frequently is largely due to the fact that a complete removal of a malignant tumor is much more difficult on account of its infiltrative growth. The presence of a recurrence in osteogenic sarcoma is not to be taken as a sign

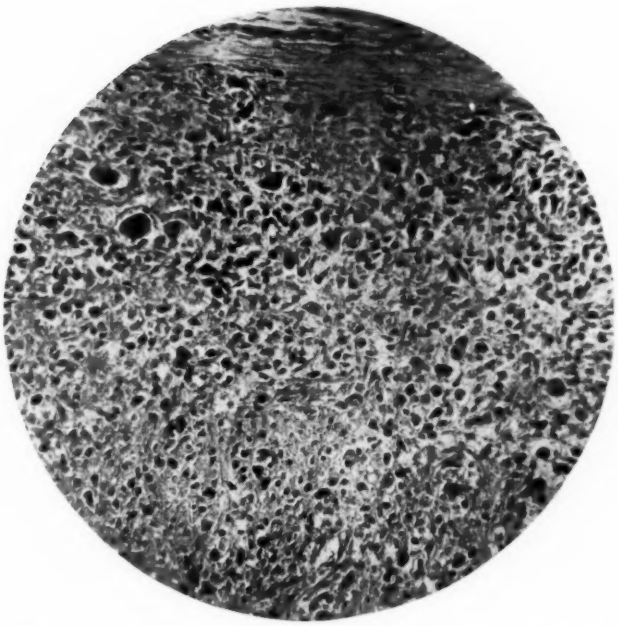


Fig. 56. Case 271. Same case as in Figure 57. Osteogenic sarcoma. Illustrating the variety of structure in different portions of the same tumor. The shown section was taken from an area close to the periosteum.

of impending death; a fatal end is imminent but the patient may go on for months. The utmost care is necessary in determining the prognosis from a recurrence if it is ulcerated; the tufts of tumor tissue as seen after exploratory incisions are usually infected and dangerous for the basing of a diagnosis or prognosis. Usually recurrences appear in osteogenic sarcoma during the first two years; however, exceptional cases are known of recurrence after a much longer interval of time. In such instances there will always be some doubt of the true nature of the growth. In the presence of metastases the prognosis must be given with reluctance; as was mentioned elsewhere, pulmonary metastases can be kept in check for a time by radiation, and even a complete disappearance of the evidence of pulmonary metastases after radiation is known to occur.

The great importance of the date of the radiological examination for the diagnosis of osteogenic sarcoma was outlined above. Attempts were made to draw conclusions from certain radiological features of osteogenic sarcoma as to the prognosis—for example, the favorable prognosis in the so-called sclerosing sarcoma, the better prognosis in osteoblastic osteogenic sarcoma as compared

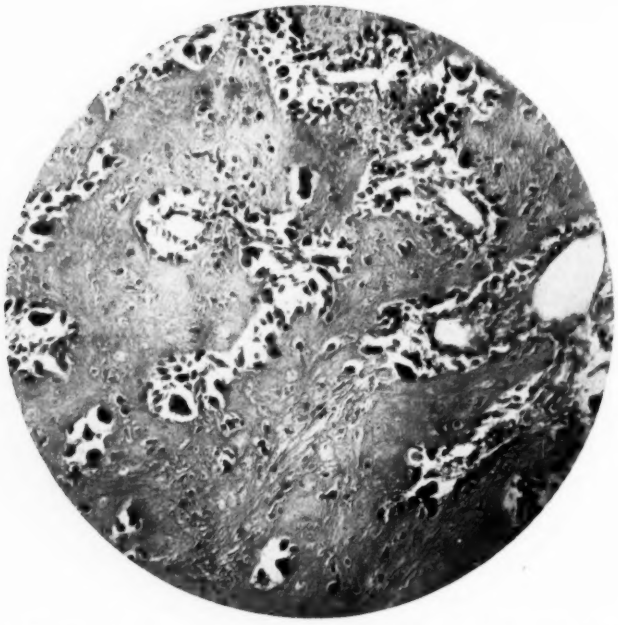


Fig. 57. Case 271. Compare with Figure 56. This section was taken from the central portion of the tumor; showing a relatively acellular hyalinized cartilaginous matrix.

with osteolytic osteogenic sarcoma when little or no ossification is seen in the roentgenogram. The utmost care is to be exercised in the prognostic evaluation of the radiological evidence. It is known how frequently a typical sclerosing osteogenic sarcoma in the roentgenogram will show numerous cellular areas; it is also generally admitted that radiation may convert a feebly osteoblastic osteogenic sarcoma into a definite osteoblastic growth in the course of several weeks. That ossification is not a dependable sign of better prognosis is evident from the fact that in the young ossification is more frequent despite the poorer prognosis than in the adult. The roentgenogram, however, is one of the most important single findings which will at once allow conclusions as to the diagnosis, stage of development, size and differentiation of the tumor.

A prognosis based upon clinical and radiological data may be greatly strengthened by findings of gross pathology and histopathology. The size of the tumor is the first factor of prognostic importance that impresses one; it is obvious that with a tumor reaching a large size without the presence of evident metastases the prognosis is better. Highly malignant bone tumors kill before they reach a large size. Encapsulation of the tumor is an important point in prognosis.

Prolonged encapsulation is a favorable sign, as very malignant osteogenic sarcomata early perforate the periosteum. Extreme vascularity of the tumor is a dangerous sign; the very vascular, so-called telangiectatic osteogenic sarcomata deserve a grave prognosis. The histological structure is an important factor in the prognosis when taken with other data, clinical, radiological, and anatomical.

The realization of the fact that the histological structure is of significance in prognosis has been greatly discredited by the assumption that all tumors may be divided in well defined groups each with an exactly defined grade of potential malignancy. The prognostic interpretation of histological findings is to be based upon the indications of the rapidity of growth of the tumor evinced by the histological structure of the tumor. The differentiation of the tumor cells is important in prognosis because an adult type of cells is usually found in the slowly growing tumors. The activity of cell division and hyperchromatism is significant as an indication of the rapidity of growth. The vascular channels and spaces lined by tumor cells characteristic of malignant sarcomata are another sign of rapidity of growth and hence of malignancy. This intimacy of tumor cells to blood vessels is important because of the readiness with which metastases may be dislodged. Necrosis if not due to radiation, is a sign of rapid growth and speaks for malignancy. Uniformity of cell structure, size and shape is a favorable sign, although pleomorphism is not an absolute indication of a grave prognosis. Tumors with a small spindle cell type have a grave prognosis.

A fact which seems to indicate a more favorable prognosis is the lymphocytic infiltration of osteogenic sarcoma. The lymphocytic infiltration was observed in 9 out of the 13 osteogenic sarcomata with five years' cure generally accepted by the Registry. The presence of the lymphocytes is not understood and involves doubt as to the diagnosis of osteogenic sarcoma in these cases; especially since the lymphocytic infiltration is rarely seen in osteogenic sarcomata which end fatally and are not incised previous to the amputation.

With the great prognostic importance of the histological structure of osteogenic sarcoma one should remember that one is not to depend entirely on the histology in deciding the prognosis if no examination of tissue from various portions of the tumor has been made; very frequently in osteogenic sarcoma one portion of the tumor differs greatly from the rest of it (Figs. 56, 57).

The few cases of osteogenic sarcoma with a five year cure in the Registry material are not sufficiently indicative to allow any general conclusions, for there is every reason to believe that the individual surgeon is more prone to register a success than a failure. On the other hand there are a large number of cases in the Registry in which the patients are still alive and have not yet reached the five year limit. Making allowances for all these arguments the very limited number of cases with a five year cure can not but greatly impress upon us the exceedingly grave prognosis of osteogenic sarcoma.

EWING'S SARCOMA

PATHOLOGY—*Gross Anatomy*

THE gross anatomy of Ewing's sarcoma is characteristic and varies from that of osteogenic sarcoma. In contrast with the latter this tumor favors the smaller bones of the extremities and the skull. When in a long pipe bone, it usually affects the shaft of the bone and not the ends. Another striking feature in which this tumor differs from osteogenic sarcoma is in the wide involvement of the shaft. The pathology of Ewing's sarcoma is less complex and easier to understand than the pathology of osteogenic sarcoma. The gross anatomic appearance in Ewing's sarcoma is mainly a result of the aggressiveness of the tumor cells, of the protective defensive measures of the affected bone, and of the regressive changes in the tumor mass. The tumor seems to begin simultaneously in numerous areas of the involved portion of the bone, in the bone-marrow filling the medullary cavity as well as the larger haversian canals. These diffusely disseminated multiple foci of tumor rapidly enlarge, become confluent, and show a great tendency to expand in all directions. The medullary cavity is frequently extensively involved at a very early stage when the cortex appears grossly intact. Fighting for space in the haversian system, the tumor diffusely dissolves the cancellous bone. The tumor cells readily spread between the bone lamellæ, which they separate and push apart. As a result of this separation of the bone lamellæ the tension and pressure, lamellæ of the involved portion of bone stand out clearly in the roentgenogram (Fig. 58). This separation of the bone lamellæ causes the bone to appear in the roentgenogram thicker than normal, as if œdematous. At a very early stage of growth of the tumor the resemblance of the bone involvement to osteomyelitis is surprising (Plate 23). The resistance of the cortical bone is soon overcome by the tumor cells which rarefy and diffusely dissolve the bone from within the numerous haversian canals. With the approach of the tumor cells to the outside of the bone the periosteum proceeds to its protective reaction. A shell of new formed bone appears as a reinforcement to the bone reduced in strength. Soon the tumor cells penetrate this advancing layer of bone and spread along its external surface. A new layer of bone is laid down by the periosteum. Shell after shell of newly produced bone is permeated by the tumor until finally the periosteum is overcome in its race with the tumor and the tumor perforates the periosteal investing capsule and begins to infiltrate

Fig. 58. Case 385. Same case as that in Plates 26 and 27. Ewing's sarcoma in a boy 8 years old. This roentgenogram was taken 1 year after the onset. Showing the separation of the tension and pressure lamellæ of the involved os calcis.

the soft tissues about the affected bone. The spreading of the tumor through the haversian system with the gradual destruction of the affected bone and the newly formed bony onion-like layers parallel with the shaft, result in the characteristic diffuse displacement of the bone by the tumor mass. To this are added the occasional foci of calcification seen in the tumor. Along with forcing its way through the bone the tumor shows a great tendency to spread up and down the shaft. The very rapid growth of the tumor in some instances prevents the periosteum from forming a true bone shell and the periosteum is pushed away by tumor tissue; when the latter has undergone necrosis it leaves a cavity running beneath the periosteum up and down the shaft. Necrotic liquefied tumor tissue occasionally resembling gross pus may fill the cavity.

The consistency of the tumor depends somewhat upon the stage of growth and the regressive changes which have taken place in it. In the very early stage of growth when the tumor is grossly limited to a relatively small area of bone the tumor presents a soft mushy white grayish substance pressed into the empty

Plate 23. Case 161. Ewing's sarcoma in a boy 13 years old. The roentgenogram and specimen show the condition 3 weeks after the onset. Notice the onion-like layers in the roentgenogram and the diffuse involvement of the bone-marrow, with a permeation of the cortex by the tumor.

spaces resulting from focal bone absorption. It resembles the appearance which one encounters in osteomyelitis with granulation tissue filling up the chambers between the necrotic bone trabecula. In later stages of growth when only traces of remaining bone are seen in the tumor tissue, the tumor represents a soft medullary crumbly growth which has extensively destroyed the shaft of the bone and infiltrated the soft parts about it. This diffuse involvement of the

Fig. 59. Case 173. Same tumor as that in Plate 31. Ewing's sarcoma. Coherent pavement sheets of cells without definite structure formation.

surrounding fascia and muscles, however, may be outlined by a distinct thin pseudocapsule, which disappears at the advanced stage of the tumor. The only supporting stroma of this very cellular brain-like tumor mass are strands of coarse connective tissue ramifying in the tumor and subdividing it into lobules.

The consistency of the tumor greatly depends upon its vascularity. While the blood supply here does not reach such extremes as in osteogenic sarcoma, still the degree of vascularity differs enough in various cases to influence the consistency of the tumor. As in other cellular tumors, regressive changes are not uncommon in Ewing's sarcoma. Necrosis in tumor areas with liquefaction may greatly resemble gross pus. In some instances such regressive changes may result in cystic formations with yellow or red jelly-like contents.

Structure

Unlike that of osteogenic sarcoma the histology of Ewing's sarcoma is simple. The type cell is small polyhedral with round oval or slightly elongated nucleus with scant clear stainless cytoplasm. The nucleus is slightly stained with scattered chromaffin granules and nucleoli which are not distinguishable or are hardly so.

Fig. 60. Case 267. Ewing's sarcoma. Showing hydropic degeneration of the tumor cells.

The cytoplasm frequently simulates a syncytium. While mitoses are abundant it is a striking feature that no tumor giant cells are seen here, the cells containing a single nucleus. The cells are extremely uniform, no pleomorphism being observed. The arrangement of the tumor cells is peculiar. Coherent pavement sheets of cells without any definite structure formation in the various sized lobules is the most frequent finding (Fig. 59). Not infrequently one encounters mucoid or hydropic degeneration of the tumor cells (Fig. 60). Complete necrosis with liquefaction is not uncommon. Frequently in section the cells are seen so close to one another as to influence the shape of the cell. A not uncommon finding is the arrangement of tumor cells about blood vessels, arterioles and capillaries (Fig. 61). An opinion has even been ventured that the blood vessels present a structural unit of Ewing's sarcoma. While the tumor cells sometimes appear arranged like a mantle about cross sections of blood capillaries, there is no evidence to support the view that, in this, Ewing's sarcoma differs from other cellular sarcomata with the so-called perithelial or perivascular arrangement. Disintegration of the tumor cells far from blood vessels leads to this appearance in any rapidly growing cellular tumor. The so-called perithelial arrangement is therefore not a necessary sign of Ewing's sarcoma, although a common one.

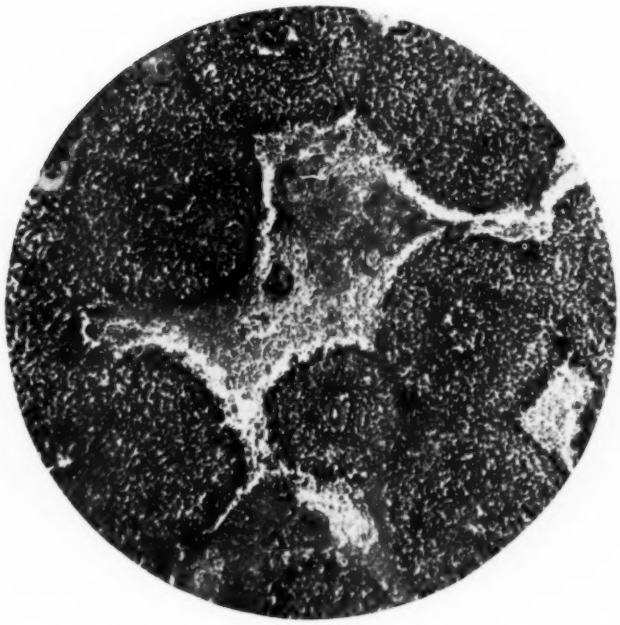


Fig. 61. Case 364. Ewing's sarcoma. Arrangement of tumor cells about arterioles and capillaries.

A striking feature of Ewing's sarcoma is the complete absence of intercellular substance. The syncytial stainless cytoplasm of the tumor cells occasionally leads one to an erroneous conclusion that the cells are embedded in an intercellular substance, but no definite cell borders can be made out there. This syncytium caused observers to suggest œdema of the tumor cells; however, the same occurrence is seen in well fixed Zenker preparations. In some spots thrombi of agglutinated red cells, and eosin staining globuli simulate intercellular matrix. Another source of error as to the presence of intercellular substance arises in connection with the hyalinized blood vessel walls supplying the tumor. Usually the eosin stained homogeneous ring of the cross section of the blood vessel wall separates the tumor cells packed around it from the lumen filled with intact blood and lined with normal endothelium (Fig. 62). In tangential longitudinal sections the blood vessel wall will readily form trabeculæ of hyalinized homogeneous connective tissue lined with rows of tumor cells (Fig. 63). These homogeneous rings and strips have been erroneously interpreted as an intercellular substance of the tumor cells. In reality these collagen fibers are not produced by the tumor cells. The tumor cells in Ewing's sarcoma do not produce anything;

Fig. 62. Case 515. Same tumor as that in Figure 63. A homogeneous ring in the cross section of the wall of the blood vessel separates the tumor cells from the lumen of the blood channel.

in this respect they seem to be far more primitive than the tumor cells in osteogenic sarcoma, if indeed any analogy may be drawn between them.

The histological picture of Ewing's sarcoma is somewhat complicated by the reaction of the tumor on the osseous tissue of the affected bone. As pointed out above, diffuse bone absorption is one of the characteristic features of Ewing's sarcoma. The extensive absorption of the shaft is accomplished without the help of giant cells. In sections one frequently sees bone trabeculæ with typical morphological features of necrotic bone closely surrounded by tumor cells. The new formed bone is of both neoplastic and metaplastic variety. Along with areas of well formed lamellary bone close to the periosteum one encounters islands of new formed bone of low grade quality in the neighborhood of the tumor cells (Fig. 64). Since the tumor cells in Ewing's sarcoma do not form any intercellular substance, this bone is doubtless a product of a regenerative process of the affected bone. This is another proof that the distinction between well formed lamellary reactive bone and trabeculated low grade tumor bone is unsound.

The typical histological structure of Ewing's sarcoma is not infrequently accompanied by peculiarities which are apt to complicate it somewhat. To these

Fig. 63. Case 515. Compare with Figure 62. Ewing's sarcoma. Showing a tangential section of the wall of a blood vessel surrounded by tumor cells. Notice the trabeculæ of the hyalinized homogeneous connective tissue of the wall.

belongs the admixture of plasma cells. This fact suggests the question of the relationship between Ewing's sarcoma and a plasma cell myeloma, a question which must be left in abeyance because of our present lack of knowledge of the origin of myelomata in general. A generous admixture of blood cells and bone marrow elements may still further complicate the appearance. As in osteogenic sarcoma one meets in Ewing's sarcoma also with an occasional lymphocytic infiltration. This infiltration when present is usually observed in the peripheral portions of the tumor where tumor frequently appears to consist of tissue truly indistinguishable from inflammation in the bone. Because this reaction is especially marked in the periphery of the tumor, one readily understands how an exploration here will lead to an erroneous diagnosis of inflammation. The admixture of these numerous cellular elements to the tumor cells often so complicates the picture that the tissue, after passing through poor technical preparations, is difficult to distinguish from myeloma or inflammatory lesions of bone.

Generalization of Ewing's sarcoma with metastatic growths elsewhere in the body proceeds through the blood and lymph stream, favoring the lymph stream

Fig. 64. Case 292. Ewing's sarcoma. Low-grade newly formed bone is seen in the close proximity of tumor cells.

more frequently than osteogenic sarcoma. Metastatic involvement of regional lymph nodes in Ewing's sarcoma is not infrequent. Unlike osteogenic sarcoma, Ewing's sarcoma is apt to metastasize to other bones of the skeleton and especially to the skull. The lungs and skull with the scalp are the most frequent seats of a metastatic Ewing's sarcoma. The metastatic foci in bone repeat all the characteristic features of Ewing's sarcoma and especially the diffuse bone absorption. The solid cellular growths occurring in skeletal and pulmonary metastases show a greater tendency to regressive changes than is the case in osteogenic sarcoma. While occasional calcification of a degenerated area in a metastatic focus has been observed, true bone formation does not occur in metastases of Ewing's sarcoma. This fact is also important proof that the origin and the physiology of the tumor cell in Ewing's sarcoma is at great variance with that of osteogenic sarcoma.

There has been noticed an attempt on the part of men defending Ewing's viewpoint of the endothelial nature of Ewing's sarcoma (Connor) to subdivide this tumor into various types. They based this subdivision upon the relation of the groups of tumor cells to the adjoining blood vessels. They describe angio-endothelioma as a variety of Ewing's sarcoma, when the tumor cells become

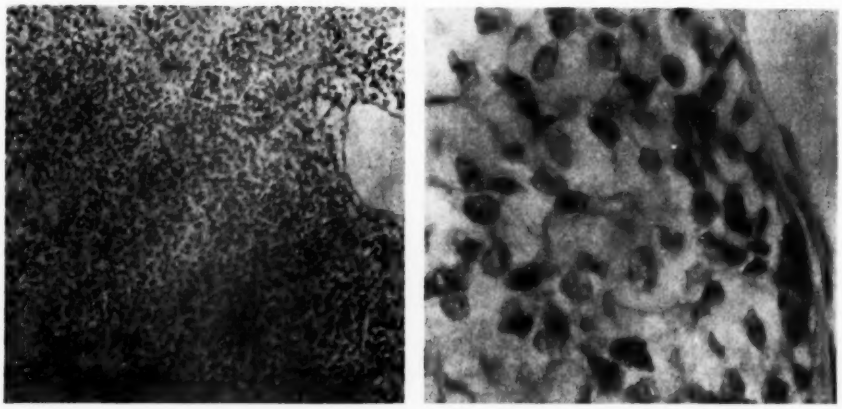


Plate 24. Case 326. Ewing's sarcoma. Showing the normal endothelium lining the blood channels surrounded by tumor cells. Low and high magnification.

flattened approaching the normal vascular endothelium and forming blood vessels and sinuses and showing a perithelial arrangement. That the so-called perithelial arrangement is not a specific feature of Ewing's sarcoma and is not infrequently observed in any rapidly growing cellular tumor has been previously stated. The presence of blood in spaces between tumor cells is no evidence that these tumor cells have been acting as a vascular unit; it is too well known with what ease a hæmorrhage may take place in a loosely arranged conglomeration of tumor cells. The attempt to interpret the tumor cells surrounding the blood cells as a vascular lining is in extreme discord with repeated statements of Ewing himself that the vascular endothelium has no relation to the origin of Ewing's sarcoma (Plate 24). An angio-endothelioma is a tumor of the vascular endothelium. It is characterized by the large cuboidal or cylindrical cells with clear cytoplasm and sharp cell membrane which constitute it. The clear cut difference between these cells and the type cell in Ewing's sarcoma is obvious. Ewing distinctly discriminates between angio-endothelioma of bone and the diffuse endothelioma (Ewing's sarcoma). Not only the histology but also the clinical behavior, the roentgenological picture and the prognosis of angio-endothelioma is different from Ewing's sarcoma. There can be no doubt that such a confusion of angio-endothelioma with Ewing's sarcoma is against all the present knowledge and evidence on hand and is misleading.

A most interesting point in the pathology of Ewing's sarcoma is the question of the origin of the tumor. While almost all who have had the opportunity to study the collection of Ewing's sarcomata in the Registry material agree that this is a specific disease, not all are willing to accept Ewing's contention that the tumor originates from the perivascular endothelium. As in any other rapidly

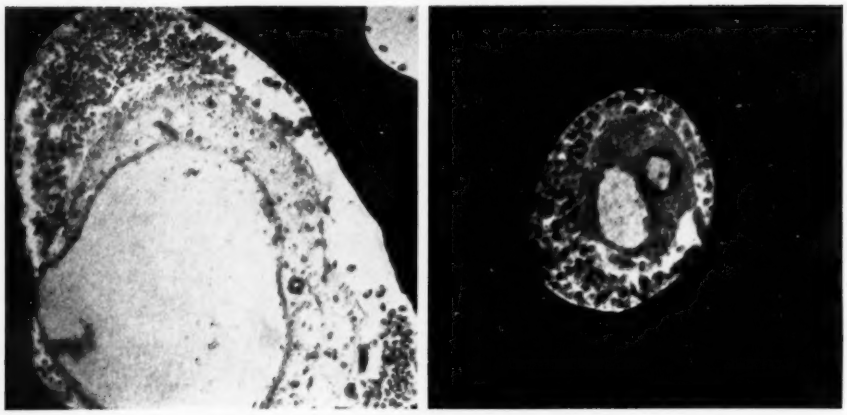


Plate 25. Case 418. Same case as that in Figure 68. Ewing's sarcoma. Minute foci of tumor cells arranged perivascularly in the haversian canals.

growing malignant tumor it is difficult to arrive at a conclusion about the origin of the tumor in the more or less advanced stages of growth, from observation alone. In the Registry material cases of Ewing's sarcoma are encountered in various stages of growth and those in the very early lesions may suggest the possible origin of the tumor. In one or two of these tumors in early growth one finds in the periphery a sprinkling of minute foci of tumor cells arranged perivascularly in the haversian canals (Plate 25). The normal presence here of perivascular lymphatics and vasa vasorum would seem to suggest that the cells originated about these anatomical structures. The nearer they are to the main tumor mass, the larger and the more numerous the foci become, and finally they merge with the main tumor mass. Whether these accumulations of tumor cells originate in the spaces where they seem to lie or whether they are brought here in the lymphatics from the main tumor, is a subject for speculation. The nature and the origin of these cells cannot be asserted positively even in these locations seemingly most favorable for study. Ewing feels that the cells of Ewing's sarcoma are "to be assigned to some endothelial category, and it may be that the interpretation of 'endothelium' may have to be widened to include this group. I do not think they arise from blood or vascular endothelium. They may possibly arise from perivascular lymphatic endothelium.[1]" Considerable argumentation of purely hypothetical character was brought in support of Ewing's hypothesis of the endothelial origin of Ewing's sarcoma. However, all these considerations are both questionable and unavailing in the absence of sufficient evidence in favor of this hypothesis. The difficulties in deciding this question of origin are great. One doubts whether morphological studies alone will ever successfully

[1]Ewing, James. Endothelial myeloma of bone. Proc. New York Path. Soc., 1924, xxiv, 93.

solve this problem. As in other tumors the essential experimental link in the chain of evidence is lacking here.

The clinical and pathological concept of the disease invites some theoretical speculations. In the face of the evidence accumulated from our study of this condition one is justified in expressing a suggestion of the probable constitutional nature of this disease. The relation of Ewing's sarcoma to lymphogenous disturbances is very suggestive. In this respect it is of significance that on the outskirts of Ewing's sarcoma there is evident an absence of blood forming elements in the bone-marrow. That Ewing's sarcoma is more than a simple bone tumor is evidenced by the diffuseness of involvement and the multiplicity of foci of origin in the same bone and also by the appearance of multiple tumors frequently very early in the course of the disease. One rarely sees a malignant tumor where the primary and secondary involvement appear almost simultaneously.

Clinical Course

The history and the clinical course of cases of Ewing's sarcoma are characteristic enough to allow a distinction between them and other bone tumors and are of great importance in the diagnosis of this disease. This fact is not yet appreciated and, as in osteogenic sarcoma, the clinical history is not given the deserved interest and attention.

The clinical incidence and frequency of Ewing's sarcoma may be judged from the fact that in a series of about 650 cases of bone tumor which have been submitted to the Registry as "bone-sarcoma" there were 40 instances of Ewing's sarcoma. If to these 40 cases one adds 10 others of more or less doubtful nature, but greatly resembling Ewing's sarcoma, one obtains the frequency of Ewing's sarcoma as 7.5 per cent of all instances diagnosed by the average operating surgeon as "bone sarcoma." In Ewing's sarcoma the male sex distinctly predominates, there being about three times as many males affected as females. In this respect Ewing's sarcoma resembles also skeletal myeloma which affects decidedly more frequently males than females. Ewing's sarcoma differs from myeloma and approaches clinically osteogenic sarcoma by the age incidence. While myeloma is encountered mostly in the fourth and fifth decades, Ewing's sarcoma falls in the last half of the first and the first half of the second decade of life. Of 40 typical Ewing's sarcomata in the Registry material 20 cases were observed in patients of the age between 6 and 15 years and only 3 cases after the age of 40. It is of interest to note that among the patients with Ewing's sarcoma some are very delicately built with slim graceful long bones, while others are fat with evidence of endocrine disturbances.

Ewing's sarcoma differs greatly from osteogenic sarcoma in location. The long pipe bones are the favored seat of the disease, but the most frequently involved bone is the tibia, next in frequency are the fibula, humerus, ulna, and

then the femur. In distinction from osteogenic sarcoma Ewing's sarcoma is a not uncommon affection of the clavicle, of the small bones of the feet, the ribs, vertebræ, mandible, and skull. The bones of the shoulder girdle and of the pelvis are all represented among these cases. The most striking feature of Ewing's sarcoma is that the site of predilection is the shaft and not the epiphyseal ends of the long bone. The involvement extends over a wide area usually more than half the length of the bone and occasionally an involvement of the entire shaft is encountered. Apparently the epiphyseal ends of a long bone are not absolutely immune to Ewing's sarcoma and occasionally one sees a Ewing's sarcoma affect the end of the bone. However a Ewing's sarcoma never proceeds as close to the end of the bone as does an osteogenic sarcoma. When a Ewing's sarcoma affects the spine, more than one vertebra is usually involved. The fact that the involved vertebræ are usually adjoining one another would seem to remove all possible doubt as to whether the involvement of all vertebræ is primary or whether some are metastases from the primary. This question of whether a multiple Ewing's sarcoma represents a true multiple primary involvement or merely metastases from one primary is even of more importance in Ewing's sarcoma than in osteogenic sarcoma. As was mentioned, a simultaneous osteogenic sarcoma of more than one bone is a curiosity, while in Ewing's sarcoma this is rather a characteristic feature of the disease (Plate 26). In the advanced stage most cases of Ewing's sarcoma reveal multiple lesions in the skeleton. Such tumors occur almost constantly in the skull and frequently in the extremities and ribs. This question is obviously of significance because of therapeutic considerations, since if the tumors are multiple there is a contra-indication to amputation while if they are metastases there is a vital indication for early aggressive radical treatment. It would seem that the extensive occurrence of skeletal tumors in advanced stages of a Ewing's sarcoma with the relative integrity of other organs points to the probability that the tumors are truly multiple and not skeletal metastases.

A frequent point in the patient's history is previous trauma at or near the seat of the tumor. The significance of trauma as an etiological factor in Ewing's sarcoma is open to the same speculations and objections as in osteogenic sarcoma, however in Ewing's sarcoma the history of trauma combined with the complaints of pain and fever, as described above, sidetracks the physician toward an erroneous diagnosis of osteomyelitis. Pain is an important and constant component of the patient's history. In distinction from pain in osteogenic sarcoma pain in Ewing's sarcoma is of a more intermittent character. Frequently the incipient pain after the trauma subsides, and the injury is entirely forgotten until an attack of sharp pain in the affected region combined with a febrile reaction reminds the patient of the trauma. The incipient attack lasts a few days and several months may pass before another attack occurs, while any

Plate 26. Case 385. Same case as that in Plate 27 and Figure 58. Ewing's sarcoma in a boy 8 years old. Death 18 months after the onset. A diffuse involvement of most of the bones of the skeleton. The tumor first arose in the left os calcis. Notice the grossly intact outline of the femur while the medullary cavity is extensively involved.

localizing symptoms may be completely absent. Each succeeding attack is of longer duration and of greater severity, and the intervals between the attacks constantly grow shorter, until finally the pain becomes of a permanent rheumatoid character. Usually at this stage a distinct tumor growth is recognized on palpation. It is not unusual to see a patient with Ewing's sarcoma up and around for more than a year, suffering from intermittent pain and attacks, before being confined to his bed. The fever accompanying the attacks may be of considerable severity. The febrile attacks throughout the course of the disease suggests that the tumor is essentially a lesion of the bone-marrow. The blood findings are of very little significance; however, not infreqeuntly it also points toward an inflammatory disturbance. The leucocytes may number 12,000 to 15,000, maintaining the normal relationship in the differential count. In far advanced cases a typical picture of secondary anæmia is present. It is easy to see how repeated attacks of fever and pain some months before tumefaction is revealed, all preceded by a trauma, lead the physician to an erroneous diagnosis of osteomyelitis. Frequently a patient with Ewing's sarcoma is operated on under a diagnosis of osteomyelitis, some are even operated on repeatedly with this diagnosis, which is also suggested by the pathologist's report of the examination of the tissue removed.

The skin overlying the bone remains free from involvement save in rare instances in far advanced stages of the disease. A surgical exploration of the tumor usually leads to a continuous ulceration with large fungous growths protruding through the incision. However a healed exploratory wound is not to be looked upon as a point against a malignant tumor since complete or temporary healing of such exploratory incisions do occur. Palpation is an important aid in the diagnosis of Ewing's sarcoma. Palpation allows one to determine the general outline of the tumor, its wide extension along the involved bone and its consistency. Repeated palpation is of primary importance when radiation is used as a diagnostic therapeutic test; one is surprised how easily the changes in outline, size and consistency of a Ewing's sarcoma subjected to radiation therapy can be perceived by palpation. Palpation may early reveal the response of the regional lymph nodes to the disease. An enlargement of the regional lymph nodes here is of even more significance than in osteogenic sarcoma, since here this is frequently not only an inflammatory enlargement but a true metastatic involvement.

The first appearance of the tumor and its further progress are rather peculiar. As mentioned above, the first febrile attacks are of more general constitutional character and no localizing is present. The tumefaction arises suddenly with signs of local hyperæmia and inflammation. After the febrile attack and most symptoms of general malaise subside, a tumor is discovered. At commencement the tumor may be slow in progress; it may also entirely subside and disappear,

merely to return with a subsequent attack in a considerably larger size. Then the growth becomes continuous and increases rapidly in size before the termination. The incipient temporary variations in size of the tumor often lead the patient and not uncommonly also the physician to an erroneous conclusion of a spontaneous cure. The patient keeps up his daily work until a new attack finally leads to a persistent tumor. Pathological fractures are not typical of Ewing's sarcoma, save in the terminal stage of the disease. Joint involvement is unknown in Ewing's sarcoma for obvious reasons: that the shaft is involved instead of the ends of the bone and that Ewing's sarcoma does not proceed to the articular cartilage.

The terminal stage of Ewing's sarcoma is different from that in osteogenic sarcoma, although it is necessary to admit that our knowledge of the final stage of the disease is much less accurate. As a general rule the patient is taken home before termination where he remains until death; necropsies of such patients are scarce. The generalization period cannot be set accurately since metastases are apt to show up on one hand in such a short interval of time as a few months after the appearance of the primary lesion, and on the other hand years may pass before metastases are found. The advent of metastases is frequently accompanied by severe febrile disturbances, especially in pulmonary involvement. Frequently the first organ to show a metastatic involvement is the skull. After the appearance of the first secondary tumor, the disease may again become stationary for some period of time especially after radiation. Months or even a year may pass before pulmonary metastases reveal themselves and secondary tumors in other bones appear. The patients are usually greatly emaciated at this stage of the disease and soon die. Metastases to the regional lymph nodes are not uncommon in Ewing's sarcoma although they are far behind the skull and the lungs in frequency. From the scarcity of evidence which it has been possible to obtain at necropsy, it seems that metastases to other soft organs are not frequent. In several cases an extensive involvement of the kidneys, liver, and spleen were observed (Plate 27).

Summarizing the average history and clinical course of a patient with Ewing's sarcoma, the most striking features are—trauma followed by intermittent pain of long duration, tumefaction, and diagnosis of osteomyelitis. When in a young person a seemingly trivial trauma is followed by an insidious onset of fever and intermittent limping due to a recurrent aching bone and is accompanied by a slight or no increase in leucocytes a tumor is more probable than is osteomyelitis. The following is a history of Case 538 of the Registry, unanimously accepted as Ewing's sarcoma. The history is a good illustration of a typical course of Ewing's sarcoma and also of the errors in diagnosis which are apt to accompany this tumor. Only by realizing how easy it is to make similar errors in this disease can such errors be avoided.

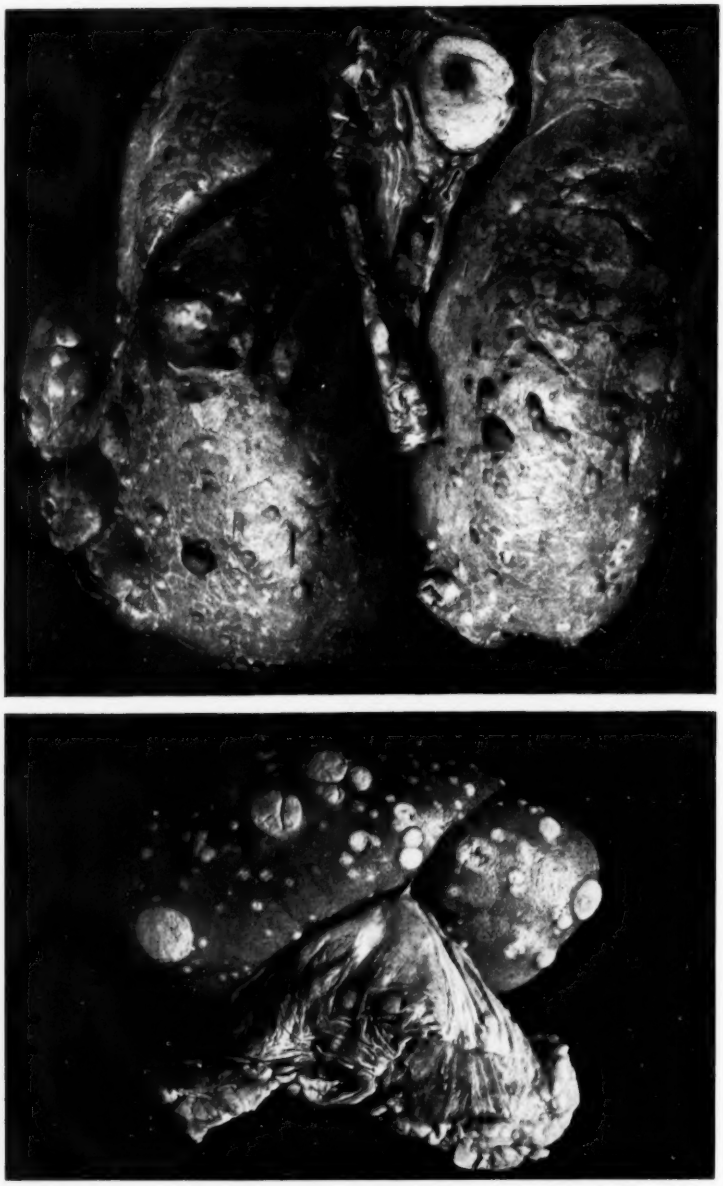


Plate 27. Case 385. See Figure 58 and Plate 26. Ewing's sarcoma. Visceral metastases.

Case 538. A boy 6 years old sustained an injury to the left leg below the knee by falling down steps. He limped and complained of pain for about a week after the accident, with fever at night. Ten days after the trauma he was seen by a physician; his temperature was 102 degrees F., pulse about 140, and leucocytosis 12,000. A roentgenological examination showed an area of absorption along the medullary canal of the tibia about 4 inches below the proximal articular end, the bone showing a definite periosteal reaction. A diagnosis of osteomyelitis was made and the patient was operated upon immediately. A hole was chiseled into the marrow cavity and a small "pus cavity" was curetted. A silver wire drain was inserted and the incision closed. The pathologist's diagnosis was "subacute osteomyelitis; pus sterile." An uneventful convalescence followed; the incision healed and the temperature dropped to normal in about 3 days. About a month later pain returned with slight fever. An examination showed the affected area in the tibia to have increased slightly in size. Nothing was done at this time. Two months later the patient was brought back with severe pain in the leg. There was localized tenderness and swelling in the neighborhood of the old operative wound. The history at this time revealed that several days previously he began to complain of soreness and pain in the leg and of fever. He walked with perceptible limp but the knee joint showed no disability. He was readmitted to the hospital with a temperature of 100 degrees F. and leucocytosis of 15,000. At this time the roentgenogram showed the absorption in the medulla and the periosteal reaction more pronounced than before (Fig. 65). A second operation was performed. Through the old incision the hole in the bone was enlarged upward toward the tibial tubercle where a "large pus pocket was found containing yellowish-red pus." The marrow cavity was curetted radically until "hard healthy bone was found on all sides, bone cavity wiped out with phenol and alcohol." The incision was again closed with a wire drain, *in situ*. The pathologist's diagnosis was "subacute osteomyelitis, agar and broth cultures of pus showed no growth."

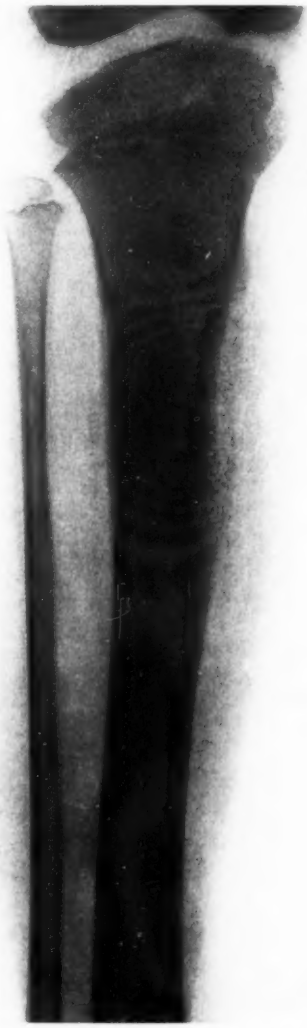


Fig. 65. Case 538. Same case as that in Plate 30. Ewing's sarcoma. The clinical history of this case is given in the text. The roentgenogram was taken before the second exploratory operation. A careful interpretation of the roentgenogram would have suggested a diagnosis of tumor at this stage while histological examination of the curettings continued to sidetrack the diagnosis for another 14 months.

About a year after the first onset of pain there was no improvement clinically in the patient's condition. The leg continued to be painful at intervals accompanied by an occasional rise in temperature. The roentgenogram at this time showed the area of destruction in the medullary canal to be about five times its size at the first operation. The neighboring bone and the medulla showed irregular deposits of calcium and an occasional area of absorption. The cortex posteriorly was partially destroyed. There was no involucrum formation. The diagnosis at this time was "osteomyelitis, probably tuberculous." Sixteen months after the first onset of pain the leg was again examined and showed no improvement clinically and a further destruction of the cortex. There was some fraying of the periosteum at the ends of the apparent involvement of the bone. Eighteen months after the first onset of pain the patient returned for roentgen-ray examination. Numerous small rarefied areas of absorption were seen throughout the bone. Plaster cast was applied. Twenty-two months after the onset of the disease the roentgenogram first raised a suspicion of malignancy. Considerable calcified material was thrown out and a soft tissue tumor surrounding the bone was seen. At this time a sinus was curetted to secure tissue for histological examination. Pathologist's report: "Typical lymphoid cell granulation tissue with one spot of tumor tissue infiltrating the granulation tissue." Following the application of a new cast a profusion of fungus-like "granulation tissue" suddenly bulged out through the window in the cast. At this time the patient was 8 years old, the duration of the illness being 2 years; he was running a septic temperature and suffering great pain. The hæmoglobin was 70 per cent, red cells numbered 2,600,000, leucocytes 9,000. The wound was again curetted for a histological diagnosis. The pathologist reported: "The diagnosis rests between sarcoma and granulation tissue; there are very cellular areas suspicious of sarcoma." Radiation therapy was started on the left leg, on the lungs, and on all other bones except the skull. Two weeks later the circumference of the left leg had become reduced from 30.25 centimeters to 27 centimeters. The granulations became of a healthy appearance; the temperature became normal, and the pain entirely disappeared. A month later radium treatment was given. The child's condition improved rapidly; he gained 3 pounds. Two months later "removal of large sequestrum was thought advisable." Sequestra were removed with a large quantity of necrotic soft tissue. Microscopically there was "no positive evidence of sarcoma." A month later after the last operation the child developed headaches; roentgenological examination of the skull showed numerous sarcomatous metastases; there was no indication of metastases in the lungs or ribs. Radiation therapy to the skull soon relieved the headaches and the general condition was fairly good. Treatment was continued for 3 months after the skull metastases were discovered, when the left leg again began to pain. The general condition of the child became poor, the granulation tissue had again piled up and acquired a foul odor, and the patient died 2 months later, 2 years and 10 months after the first onset of pain.

Diagnosis

In studying Ewing's sarcoma, one is especially impressed by the danger of an overestimation of the roentgenological method of examination on one hand and an exaggeration of the diagnostic possibilities of histological examination alone on the other. In all skeletal tumors but especially in Ewing's sarcoma if one attempts to base a diagnosis on the roentgenogram alone one will frequently be mistaken, but to satisfy oneself in the establishment of a diagnosis by a histological examination alone is not less erroneous. Only a well-planned and carefully conducted analysis of the history and clinical picture together with a study

of the findings of the radiological and pathological examination will reduce *ad minimum* the errors in diagnosis. In the presence of Ewing's sarcoma the clinical history and findings as outlined above will frequently suggest the diagnosis. Among the salient features in the clinical history of the disease, trauma has a definite place. Whatever the true causative relation of trauma to Ewing's sarcoma may be it is significant for diagnosis that trauma is commonly present in the history. Next in succession to trauma are the intermittent symptoms, with apparent complete disappearance of trouble merely to return in a more severe form.

As a rule the periodically aching bone, historically following after a trauma, together with a general constitutional response, leads to a diagnosis of osteomyelitis. This error is so frequent that it is in itself a characteristic feature of Ewing's sarcoma. The findings of the subsequent early radiological examination are not incompatible with osteomyelitis. This diagnosis may be supported by the gross appearance and frequently by the histological examination of "granulations" removed during an attempt at curettage and drainage of an osteomyelitic process. Finally, after the surgical intervention the intermittently progressing tumor begins to grow rapidly and soon the error in diagnosis is exposed. The fact that osteomyelitis is almost a typical error in diagnosing Ewing's sarcoma is not surprising when one realizes how much in common these two conditions have in a superficial examination. The triad of clinical symptoms of Ewing's sarcoma, the history of trauma, intermittent pain in a bone, and the signs of inflammation greatly resemble the triad of osteomyelitis,—intermittent boils, trauma, and pain in the bone. The slow progress of the disease and the usually not very prominent constitutional reaction suggest a chronic, slowly proceeding osteomyelitis; an acute inflammation is rarely considered. In this respect Ewing's sarcoma differs from osteogenic sarcoma when an acute or subacute osteomyelitis is not infrequently erroneously considered. The not uncommon sudden onset of pain in osteogenic sarcoma accompanied as it may be by inflammation of the skin, fluctuation or pseudofluctuation and fever is a satisfactory explanation of this divergence between Ewing's sarcoma and osteogenic sarcoma. The frequency of mistaking a malignant bone tumor for osteomyelitis suggests that no diagnosis of a chronic inflammatory skeletal process should be made before the probability of a malignant tumor is considered and ruled out. On the other hand it is not unusual to observe an erroneous consideration of osteomyelitis as Ewing's sarcoma or osteogenic sarcoma. Cases are known in which radical surgery led to amputation of a limb because of such mistaking of osteomyelitis for a malignant tumor. Such errors were more frequent before modern diagnostic radiology was known. Especially suspicious in its resemblance to osteolytic osteogenic sarcoma is a central osteomyelitic abscess, so-called Brodie's abscess; its usual location in the metaphysis is not incompatible

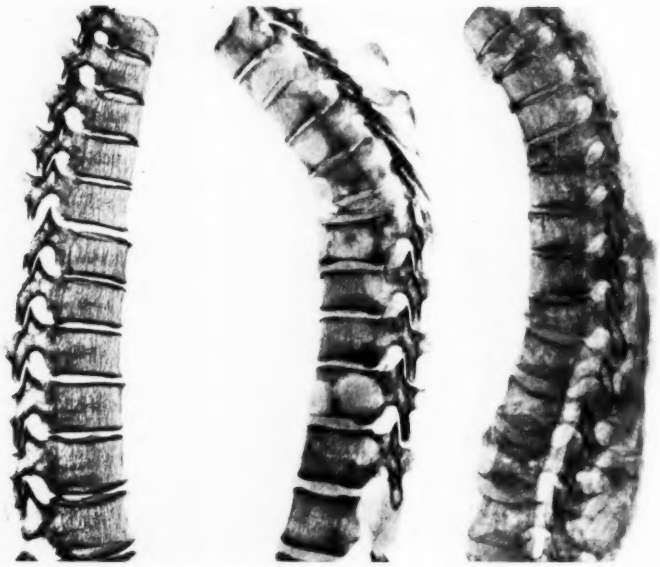


Plate 28. (1) Normal spine, (2) metastases of a carcinoma of the breast, (3) myeloma of the spine.

with osteogenic sarcoma. However in the roentgenogram it differs from osteogenic sarcoma in the sclerosing osteitis and periostitis surrounding the lesion, features absent in osteogenic sarcoma.

A tuberculous skeletal involvement is considered chiefly in tumors of the spine. Here mostly Ewing's sarcoma is concerned, because of the favorite seat of Ewing's sarcoma in the vertebræ. A careful analysis of the clinical findings will greatly help a differentiation. Thus sensory and motor disturbances are a much earlier complication in Ewing's sarcoma than in tuberculous spondylitis. The absence of cold abscesses in advanced stages of the disease also speaks for tumor. Another differentiating point has been described in the literature: Œdema of the soft tissues overlying a vertebra which is the seat of a malignant tumor. Most important for the differentiation is the roentgenogram. It should be realized, however, that it will be disappointing to expect findings which would warrant a diagnosis from a large film where most of the spine is exposed. The suspected area of the spine, not more than 3 to 5 vertebræ, should be exposed on one film so that they all lie in the direction of the central rays.

Experience has shown that interpretation of roentgenograms of the spine are little understood by the average clinician; the fundamentals of the normal appearance of the spine in the roentgenogram are not generally known. In a roentgenogram of a normal vertebra one notices two systems of bone trabeculæ

which intersect perpendicularly and which are interrupted in their continuity by a sagittal canal carrying the main central vein of the vertebra. Most important is the fact that the height of each caudally following vertebra is larger than, or at least of the same size as, that of the one above it. A longitudinal diameter of a vertebra shorter than the corresponding diameter of the vertebra above it is to be considered as absolute indication of a pathological involvement of the vertebra. It is hardly necessary to say that the integrity of the sharpness of the outline and the uniformity of the density of a vertebra is as important diagnostically as in any other bone (Plate 28).

An important clinical symptom frequently neglected by the physician is sciatica. Sciatica may be the first symptom in tumors of the lumbar and sacral spine and the bones of the pelvis. It is also encountered occasionally in tumors of the cord as well as tumors of the soft organs of the pelvis. It is significant that with an increase of the experience of the surgeon the incidence of idiopathic sciatica decreases.

Fig. 66. Case 267. Ewing's sarcoma in a boy 8 years old. Amputation and later application of radium to abdominal metastases (?). The patient is well 5 years after the onset. The roentgenogram was taken 5 months after the onset. The apparent widening of the shaft and the displacement of the old bone by the tumor with the extensive involvement of the shaft is typical of Ewing's sarcoma.

The differentiation of Ewing's sarcoma from osteogenic sarcoma is accompanied with difficulties. Much reliance can be placed upon the clinical history and course, but the roentgenogram is frequently so characteristic in Ewing's sarcoma that it decides the diagnosis. A roentgenogram of an early Ewing's sarcoma will show a diffuse involvement of the bone; this is probably due to a diffuse origin of the tumor throughout the haversian system. Usually the diaphysis is involved and not the ends. Of 40 cases of Ewing's sarcoma,

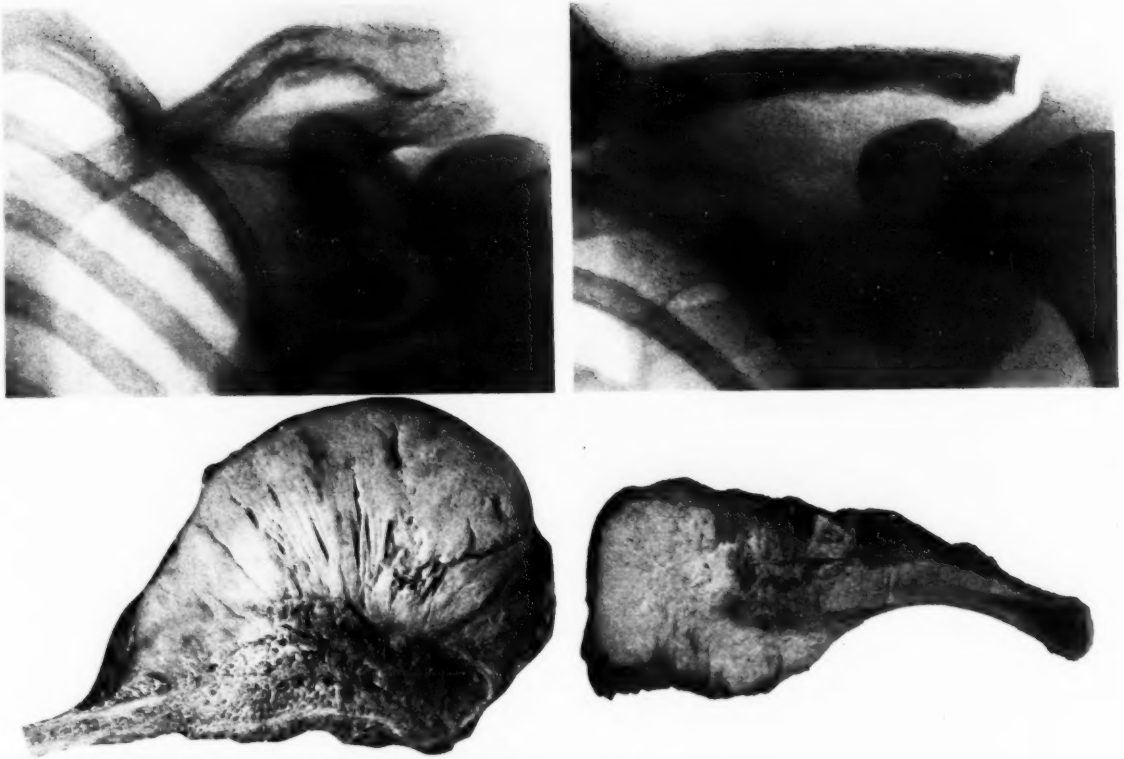


Plate 29. Cases 162 and 504. Roentgenogram and photograph of each specimen:
Lower left, osteogenic sarcoma in a girl 11 years old; 4 months after the onset. Death 11 months after the onset.
Lower right, Ewing's sarcoma in a woman 23 years old; 3 months after the onset. Death 8 months after the onset.
Showing the difference between osteogenic sarcoma and Ewing's sarcoma. In Ewing's sarcoma a destructive roughening of the periosteum is seen; the shaft of the bone is widened, while in osteogenic sarcoma the shaft with a preserved outline is passing through the tumor mass. In the photograph, Ewing's sarcoma shows a bulky fleshy soft tumor mass, while in osteogenic sarcoma the tumor mass is supported by a skeleton of radiating spicules. The periosteal spindle is well illustrated in osteogenic sarcoma.

in only two the ends of the bone were the seat of the tumor. The involvement is wide; commonly about half of the length of the bone is involved. Unlike osteogenic sarcoma which in the roentgenogram shows the shaft passing through the tumor mass grossly intact, Ewing's sarcoma shows the bone being slowly displaced by the tumor tissue (Fig. 66). The smooth absorption of the bone is accompanied by a widening of the shaft with frayed longitudinal bone trabeculæ. A destructive roughening of the periosteum is observed but there is no periosteal spindle and no lipping of the periosteum (Plate 29). The new bone is laid down parallel to the shaft; only in 3 cases out of 40 a radiating arrangement of new bone was observed (Figs. 67 and 68). In macerated and dried specimens small radiating spicules may be occasionally seen while in the roentgenogram they could not be perceived. The metastases in bone, which are typical of Ewing's sarcoma, have an appearance similar to the primary growth in the roentgenogram. A differentiation between Ewing's sarcoma and metastatic growths will be easily made if one remembers that metastases occur in the very young or after the age of 40, while most Ewing's sarcomata are seen in early adolescence.

In Ewing's sarcoma the pathological examination of tissue removed at biopsies is less reliable than in osteogenic sarcoma. If, in osteogenic sarcoma, tissue obtained through biopsies is not representative enough to allow a correct diagnosis, even more is this the case with Ewing's sarcoma. Also the fallacies of technical procedure are such that one rarely encounters a well prepared histological section which would warrant a diagnosis for the pathologist non-expert in bone tumors. There are numerous examples of the pathologist supporting the erroneous diagnosis made clinically and roentgenologically of osteomyelitis in the presence of Ewing's sarcoma. On the other hand it is fully attested at present that no other skeletal lesion so rapidly responds to radiation as Ewing's sarcoma. The suppression of Ewing's sarcoma by radiation is so marked that it in itself presents a most valuable diagnostic point in this disease. Viewed largely, the diagnostic limitations of pathology combined with the dangers of exploratory incision in Ewing's sarcoma require that a therapeutic radiation test should be substituted for exploratory incisions in cases suspected of Ewing's sarcoma (Plate 30). The intermittent symptoms, the characteristic roentgenogram, the therapeutic result will lead to a correct diagnosis.

Therapy and Prognosis

The general points of the therapy of bone sarcoma as pointed out in the chapter on osteogenic sarcoma are partly applicable also to Ewing's sarcoma. The general rapid response to radiation of Ewing's sarcoma is the main difference between this tumor and osteogenic sarcoma. Heavy radium and roentgen-ray radiation suppresses Ewing's sarcoma and causes a regression of the tumor in a short time (Plates 31 and 32). Unfortunately the enthusiasm from the first

while in osteogenic sarcoma the tumor mass is supported by a skeleton of radiating spicules. The periosteal spindle is well illustrated in osteogenic sarcoma.

Fig. 67. Case 541. Ewing's sarcoma in a boy of 12 years. The roentgenogram was taken 1 month after the onset of the disease. The widening of the shaft with the parallel striations is typical of Ewing's sarcoma.

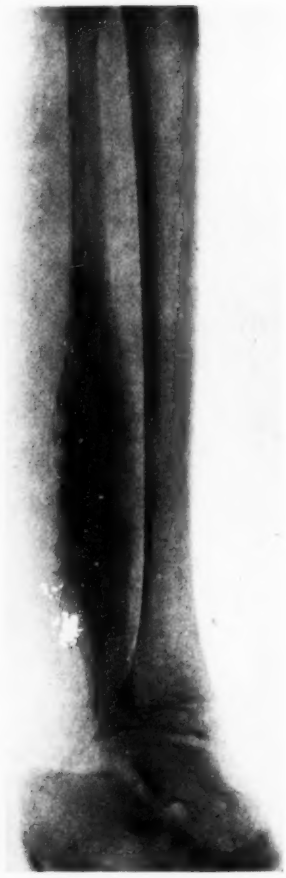


Fig. 68. Case 418. Same case as that in Plate 25. Ewing's sarcoma in a boy 10 years old. Notice the radiating spicules of the new bone in the presence of all features of Ewing's sarcoma in the roentgenogram.

impression lost ground later when experience showed that the tumor, being very susceptible to physical therapy, tends to recur and slowly become refractory to further radiation. There are, however, at present on record cases of Ewing's sarcoma remaining well up to four years after the onset, in which radiation alone was used. In the material of the Registry the patients with Ewing's sarcoma lived longer who had operative treatment accompanied by radiation.

Plate 30. Case 538. The same case as that in Figure 65. Ewing's sarcoma. Showing the response to radiation therapy. Roentgenograms taken 16 months after the onset, (left) and after several exposures to roentgen rays. By a trial of radiation at a much earlier date one would have avoided in this case repeated exploratory operations.

Plate 31. Case 173. Same case as that in Figure 59. Ewing's sarcoma in a boy 16 years old. Roentgenogram taken 8 months after the onset; the leg was amputated 4 years after the onset. In the interim radium was used. Showing the temporary suppression of growth of the tumor by radiation; the gross outline of the cortex is almost completely restored.

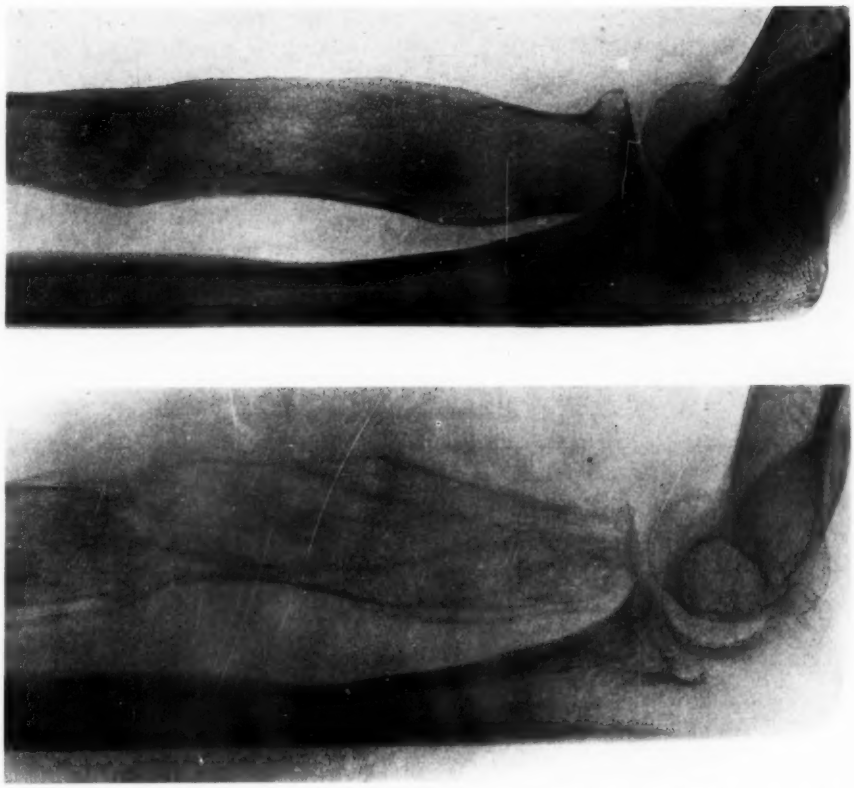


Plate 32. Case 352. Ewing's sarcoma in a girl 14 years old. An exploration was made about 1 year after the onset, at which time radium packs were applied with a good immediate local result. Death with skull metastases 3 years and 2 months after the onset.

Yet since radical surgery does not seem to prevent multiple tumors in the skeleton, amputation is possibly futile. The best way probably to deal with a Ewing's sarcoma is first immobilization of the affected region and heavy radiation; when the tumor recurs locally, then excision or even amputation can be performed. Radiation is to be continued for a longer period of time than is usually done in osteogenic sarcoma. Cases are known of the spontaneous disappearance of roentgenological evidence of pulmonary metastases for a period of years after heavy radiation. Overradiation is to be feared, since it prevents further radiation because of skin burns and because it is apt to destroy the bone produced as a result of the previous radiation. The dosage of radiation required is a matter of experience. The general impression is that radium is to be preferred to roentgen ray in Ewing's sarcoma.

As with all malignant bone tumors, prognosis as to life expectancy in Ewing's sarcoma is not possible for reasons which were pointed out elsewhere. In Ewing's sarcoma the treatment especially intimately influences the prognosis. If an average prognosis of cases with a diverse clinical course, seen in different stages of development and treated by varying methods is of any importance, then the average duration of life in Ewing's sarcoma is longer than in osteogenic sarcoma—in Ewing's sarcoma about three years while in osteogenic sarcoma about twenty months. The wide range of life duration is evidenced by the fact that one patient of the Registry cases is alive sixteen years after the onset of the disease while several patients died 5 months after the onset of the tumor.

As a disease favoring the young and adolescent Ewing's sarcoma does not show any appreciable difference in prognosis in relation to the age of the patient. There is also no evidence as yet to lead to the presumption that histological varieties of Ewing's sarcoma have anything to do with the prognosis, and the attempts made (Connor) to segregate certain varieties of these tumors probably lack clinical justification. Because of the longer life duration in Ewing's sarcoma there is greater possibility of influencing the tumor by radiation and occasional surgery, and a longer period of time in which to do it.

MYELOMA

PATHOLOGY—*Gross Anatomy*

THE voluminous and detailed descriptions in the literature of this disease are frequently based upon questionable material. As a matter of fact, myeloma is a rare condition, experienced pathologists having seen but few cases in years. Not infrequently a tumor is diagnosed as myeloma simply because of the round shape of the cells composing it. That Ewing's sarcoma is sometimes confused with and taken for myeloma I have repeatedly observed. These facts partially explain the reasons why the pathology and also the clinical course of myeloma is often being presented in different, frequently contradicting descriptions. Probably this will continue to exist until we learn more of the normal anatomy of the hæmatopoietic system in general. The numerous names offered by various investigators for the disease known today as myeloma attest the great confusion existing as to the true nature of the condition. Although myelomata are rare, they are of sufficiently frequent occurrence to warrant a consideration of this tumor in a study of primary malignant bone tumors.

In divergence with osteogenic sarcoma myelomata favor the midportions of the long bones, especially the humerus and the femur and the flat bones of the skeleton such as the sternum, ribs, skull, and the vertebræ. The tumor forms nodules ranging in size between that of a bean and that of an orange. It readily spreads along the marrow cavity, replacing the normal bone-marrow so that only on the edges of the expanding growth can one see tumor cells intermingling with normal bone-marrow elements. The tumor steadily expands in the transverse diameter of the bone. The destruction of the bone is less diffuse, is of more rapid course, and of more circumscribed character than that seen in Ewing's sarcoma. Usually no osteoclasts are assisting the tumor cells in the destruction of the bone. The cancellous bone rapidly melts down at the approach of the tumor cells. The cortical bone becomes eroded and the tumor protrudes beneath the periosteum, which is slowly undermined and pushed away from the doomed cortex. Repeatedly the periosteum forms new layers of bone in place of the thinned out or completely destroyed cortex. This new bone shell, however, rapidly melts down under the aggression of the tumor cells. As a result of this periosteal reaction the bone cortex seems expanded and the medullary cavity widened. Finally the periosteum becomes perforated here and there and the

Plate 33. Case 461. Myeloma. The medullary cavity is stuffed with tumor; pathological fracture; hæmorrhage and blood cysts.

tumor infiltrates the surrounding structures. Bending and deformity of the involved weight bearing bone may take place and spontaneous infraction or complete fracture may follow. The gross specimen usually shows a greatly thinned cortex of an expanded bone filled with an increased medullary substance, soft or firm, translucent or opaque with little or no bone trabeculæ in it. The tumor mass is gray, reddish gray, or deep red according to the vascularity. The

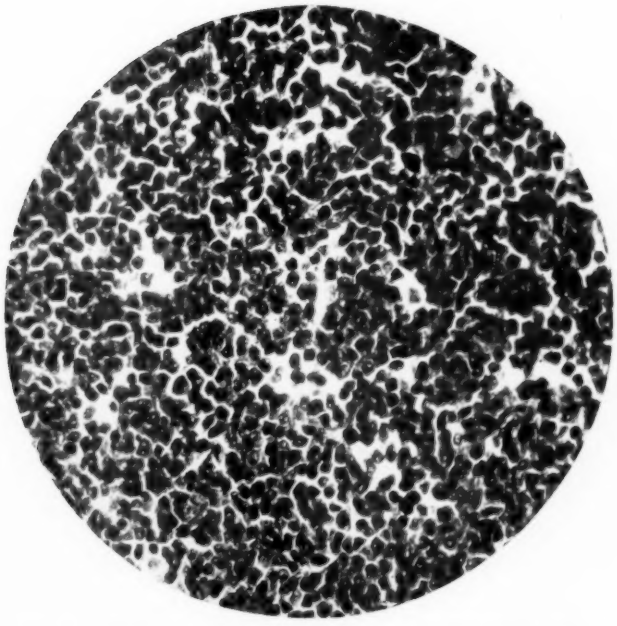


Fig. 69. Case 242. Compare with Figure 70. Myeloma, plasmocytoma. Low magnification.

vascularity is sometimes so great as to cause pulsation of the tumor. Hæmorrhage from the very thin walled capillaries is frequent; hence blood cysts and pigmentation. Extensive regressive changes are frequent with the usual changes in the cells (Plate 33).

Structure

The histogenesis of myeloma is still a field of much theorizing and speculation. Taking myelocytes, lymphocytes, and mononucleated red blood cells as the specific bone marrow cells, tumors have been described which were said to originate from each of these basic specific cellular elements. The majority of myelomata are composed of nongranulated mononuclear cells with opaque basophilic protoplasm, closely resembling the plasma cells, with which they are frequently identified. In other words the most frequent myelomata seem to be constituted by cells the origin of which is still being debated. In connection with this, it may be mentioned that an idea that all types of myeloma are composed of cells merely representing various stages of anaplasia in a single cell is being entertained. Ewing thinks that the fact that the myelocytoma is of more active growth than the plasmo cytoma, suggests that a greater degree of anaplasia may

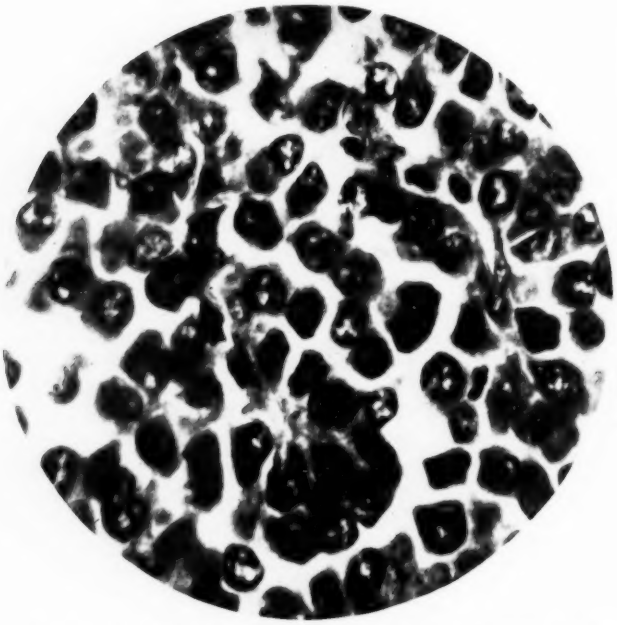


Fig. 70. Case 242. Compare with Figure 69. Plasmocytoma. High magnification.

lead a plasma cell to become a cell resembling a myelocyte. The general trend of opinion favors the origin of the plasma cell from the lymphocyte or from endothelium. According to some authors, the ability of the plasma cell myeloma to destroy bone speaks in favor of this latter alternative. Whatever the histogenesis of myeloma may be, four various types of myeloma are classified by various authors each with a different type cell and possibly with a slightly different clinical course. In order of frequency of occurrence these types are plasmocytoma, lymphocytoma, myelocytoma, and erythroblastoma. The type cell in each of these types is easily recognized in sections, but the rapidity of growth and frequent degenerative changes may obscure the appearance sufficiently to make it extremely difficult to differentiate exactly the type cell of the tumor. Among the plasmocytomata one encounters a small and large cell variety; the latter are said to be of more rapid growth and somewhat worse prognosis. The cells are usually round and oval but from pressure may become polygonal and even spindle formed (Figs. 69, 70). The nuclei are small and eccentric with granules of chromatin arranged along the nucleolar membrane. Not uncommonly the cells are multinucleated. The tumor cells are loosely packed, without any intercellular substance, without any definite arrangement and with

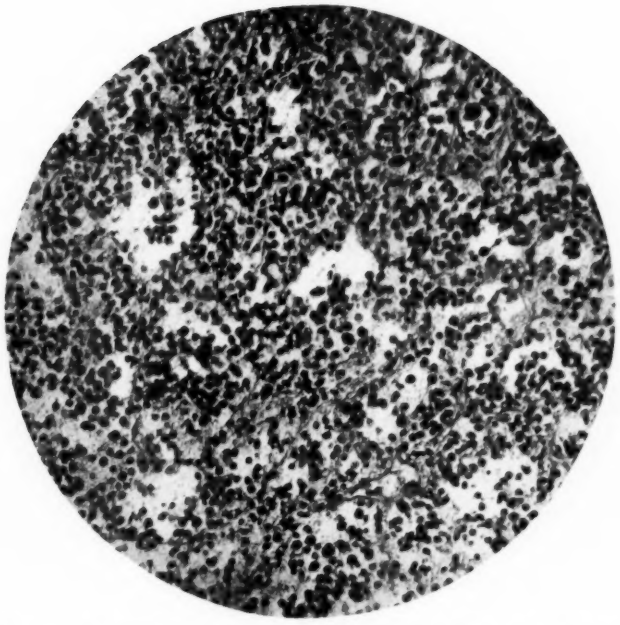


Fig. 71. Case 517. Myeloma, myelocytoma; a capillary web variety.

a scanty stroma supporting them. The lymphocytoma is less aggressive. It is composed of typical small lymphocytes. The type cell in myelocytoma resembles a myelocyte (Fig. 71). The large hyperchromatic cell with prominent granules readily suggests the myelomatous nature of the tumor. The erythroblastoma is called so because the two cases described in the literature showed hæmoglobin in the tumor cells. Hæmoglobin was so abundant that it caused a deeply brownish red color of the tumor. The Registry material is poor in myeloma, the 9 typical and 5 questionable cases registered belonging to neither of the two last mentioned types. For common use among clinicians and pathologists a subdivision of myeloma into types is probably of no advantage. Distinction of finer points in the histology of myeloma is at present of purely scientific interest.

Clinical Course

Unlike osteogenic sarcoma and Ewing's sarcoma neither the history nor the physical examination of a patient with myeloma in the early stage is in any way characteristic of the disease. This fact combined with the rare incidence of myeloma largely explains the fact that myeloma is seldom recognized before very extensive involvement takes place. The disease chiefly affects males mostly

Fig. 72. Courtesy of Dr. Holmes, Massachusetts General Hospital; not registered. Myeloma in a woman 56 years old. The same case as that in Figure 73.

between the ages of 40 and 60. Myeloma in the advanced stages extends throughout most of the bone, not infrequently replacing the entire bone marrow. For a long time it was considered a multiple lesion, originating and developing in multiple foci. Only one case of solitary myeloma was recorded.

Despite the multiplicity of myeloma in most cases the disease is connected with a traumatic history. As in other malignant bone tumors, pain is an important factor in the clinical course of myeloma, although it seems to appear considerably later than in most osteogenic sarcomata. In myeloma of the spine, cord pressure is a frequent finding. The general condition of the patient depends upon the stage of the disease; in far advanced stages a peculiar cachexia and emaciation is seen. A secondary anæmia as a result of a marked destruction of the bone marrow accompanies the cachexia. In early stages the blood may not show any changes. Cases have been described of an increase of myelocytes or plasma cells in the blood. The incipient fever is later displaced by a subnormal temperature. Palpation may sometimes reveal crepitation of the extremely thin cortex, an enlarged spleen and possibly a hyperplasia of the regional lymph nodes. The enlargement of the spleen is possibly compensatory to the destruction of the bone marrow or in the advanced stages is perhaps due to toxæmia.

Because of the rapidity of bone destruction, a pathological fracture, a bone deformity or a crushing of a vertebral body with a paraplegia is frequently the first sign of the bone lesion. Generalization of the disease through metastasizing to other organs is a feature of the natural life of a myeloma. However, frequently cases come to postmortem examination without metastases to soft organs. This is probably because the patients die early from the severe emaciation and intoxication as a result of extensive bone-marrow destruction.

Fig. 73. Same case as shown in Figure 72. Myeloma in a woman 56 years old.

Secondary growths have been seen in the liver, spleen, kidneys, regional lymph nodes, and skin.

Diagnosis

The radiological method is the main one in the diagnosing of myeloma. The history and clinical picture of an early stage of the disease is not remarkable as far as diagnostic possibilities are concerned except in regard to multiplicity and situation. In a far advanced case the multiplicity of involvement suggests multiple myeloma rather than Ewing's sarcoma. The absence of pulmonary metastases in the presence of very extensive skeletal involvement is the rule in myeloma. The age of the patient is also suggestive of myeloma; they occur after 40 years of age. Albumosuria and the presence of Bence-Jones proteins in the urine which for a long time has been considered pathognomonic for myeloma, may be absent in a large percentage of cases—according to some observers in one half. On the other hand in occasional carcinomatous skeletal metastases and other medullary tumors the presence of Bence-Jones proteins has been observed.

For obvious reasons morphological blood studies, which, as a routine, should be made in all skeletal tumors, should never be omitted in myeloma. In the roentgenogram myeloma reveals well outlined, definitely circumscribed areas of diminished density. At one stage or other these areas may be large and single or small and multiple in the same bone spreading all along the medullary cavity. The bone is slightly expanded by the individual nodules so that the extremely thinned cortex is irregularly wavy in outline and not uniform in thickness (Figs. 72, 73). There may be minute nodules in the cortex itself. A perforation of the cortex is rare and so is an infiltration of the soft tissues about the involved bone. The very characteristic roentgenogram may be greatly obscured by frequent infractions or complete pathological fractures, which may present diagnostic difficulties. When myeloma is located in the spine it is difficult to identify the individual myelomatous nodules because of the greatly atrophied bone structure. Partial collapse of a vertebra and apparent disappearance of intervertebral discs is not an infrequent occurrence in myeloma of the spine. A radiological examination of the whole skeleton is extremely important.

In making a diagnosis from a histological section one has to keep in mind that an overstaining with hæmatoxylin may lead to mistaking Ewing's sarcoma for myeloma. Myeloma generally shows mild pleomorphism as compared to Ewing's sarcoma. An exploratory incision is both undesirable and unnecessary because of the promptness with which myeloma responds to radiation. The diagnosis is early established from the very characteristic roentgenogram.

Therapy and Prognosis

As a tumor composed of lymphoid cellular elements, myeloma is readily affected by radium and high voltage roentgen rays. The local growths disappear after repeated radiation, and pathological fractures may unite under such treatment, but ultimately death follows from extension and metastases. When the disease appears with multiple tumors there obviously can be no choice between surgical treatment and radiation, the latter being the sole recourse. In the rare instances of solitary myeloma, amputation may be justified and according to some observers it may for long postpone or even prevent metastases. However, the frequent location of myeloma in the spine and the sternum will decide against surgical treatment even in cases of solitary tumors. The histological variations of the tumor do not influence the effect from radiation. It is questionable, however, whether continuation of radiation is permissible when the leucopenia reaches 2,500. To prevent a pathological fracture, immobilization of the involved limb and recumbency is essential. On the whole myeloma may prove occasionally of decidedly better prognosis than other malignant bone tumors. Cases are on record in which ten and more years have passed between the clinical onset and the fatal termination of the disease.

UNCLASSIFIED SARCOMA

IN the group of "unclassified sarcoma" are included, aside from a few atypical instances, two conditions which are endowed with a typical anatomical structure and characteristic clinical behavior. The exceptional rarity of these conditions has prompted me to refrain from a segregation of them under separate headings in the classification. These tumors are angio-endothelioma and extraperiosteal sarcoma of bone.

ANGIO-ENDOTHELIOMA

Pathology

OUR knowledge of osteogenic sarcoma, Ewing's sarcoma, and giant cell tumor has gained tremendously from the Registry, but the latter has contributed little to the knowledge of angio-endothelioma. The pathology of angio-endothelioma of bone is still a very confused problem. The reasons are numerous. Since the days when Golgi, over fifty years ago, first offered the name "endothelioma" this term has been applied extensively to uncommon tumors both of soft tissues and of the skeleton. The at first sight histological resemblance of endothelioma to carcinoma has led to the diagnosis of endothelioma in cases of secondary carcinoma in which one failed to find the primary tumor. This error has been followed in our days by the tendency of pathologists to dodge the diagnosis of endothelioma and to substitute for it carcinoma even in cases in which much of the histological and clinical evidence is in favor of the former diagnosis. Further confusion was brought in by the fact that in the literature the rare primary angio-endothelioma of bone is confounded with an occasional secondary involvement of the bone by an endothelioma of the adjoining soft tissues. Such lesions are especially frequent in the skull where the endothelium lined dura represents the endosteum of the bones of the skull. The local changes in the skull overlying a dural endothelioma or meningioma, as Cushing calls this tumor, lead to the formation of a dome shaped bony prominence, which on section shows hyperostosis infiltrated by tumor elements from the underlying primary tumor of the dura. Often these changes in the skull have been mistaken for sarcoma, and in such cases the primary lesion, the tumor of the dura, was overlooked. It is easy to see how such an error can be made when it is realized

Fig. 74. Case 154. Same case as that in Figure 75. Skeletal angio-endothelioma. The tumor cells form alveoli and tubuli.

that the fan-like radiation structure of the hyperostosis in the roentgenogram is reminiscent of the structure frequently seen in osteogenic sarcoma. It is hardly necessary to point out here the dangers, on one hand, of mistaking a secondary tumor of the bone for a primary and, on the other hand, of confusing different types of tumor which have nothing in common clinically or anatomically. It is fully ascertained that endothelioma of the dura does not metastasize and is not even locally malignant as far as infiltration of the brain is concerned.

Another source of error arose in connection with the facts that microscopically one of the leading features of angio-endothelioma is the abundant vascularity; a confusion of angio-endothelioma with angioma and angiosarcoma is probably best explained by this fact. However this may be, this still further contributed to the confusion existing about angio-endothelioma of bone. Various interpretations by individual investigators of histological findings even in true angio-endothelioma of bone did not help much to clear this problem. Such a discrepancy of views and opinions is only natural when one realizes that the tumor is exceedingly rare, the men with most experience having seen only a few cases.

The knowledge of the embryological relations of the cells called endothelium is yet far from complete. However, it is fully attested that the endothelium is

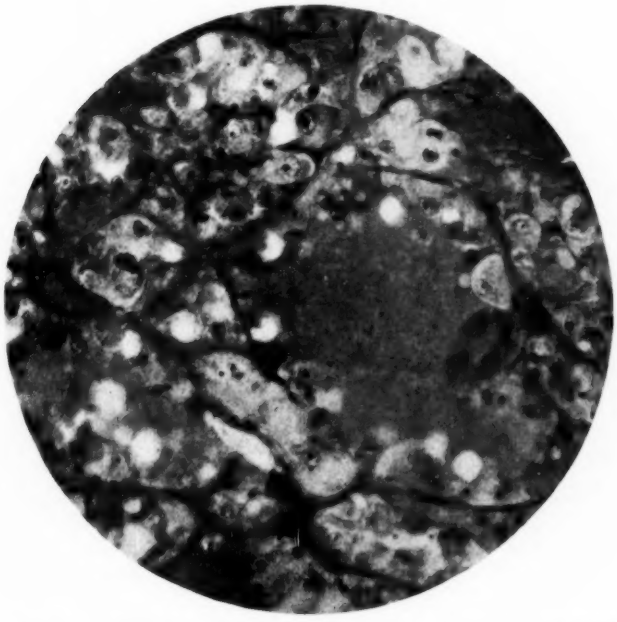


Fig. 75. Case 154. Same case as that in Figure 74. Skeletal angio-endothelioma. Alveoli with intact blood are lined by a single row of tumor cells simulating vascular endothelium.

derived from mesenchymal cells; it has differentiated along various lines because of its function—walling of vascular channels and spaces. As has been mentioned, angio-endothelioma of bone is a very rare tumor, the Registry having, in my opinion, only two cases. These two cases have been used by me in a special study with the intention of urging recognition of angio-endothelioma of bone as a definite anatomical entity.[1] It is obvious that no very extensive and definite conclusions as to their histology can and should be attempted,—it is not safe theoretically and it is of no importance from the clinical point of view. There is no sufficient evidence to support a distinction between hæm- and lymph-angio-endothelioma at least as far as bone is concerned; the numerous pitfalls surrounding such detailed studies in bone may lead to a confusion in which our meagre knowledge of angio-endothelioma of bone would be stripped of the facts at present fully attested.

In general features the most frequent histology of skeletal angio-endothelioma will at first glance impress one with the resemblance to an adenomatous growth.

[1]Kolodny, Anatole. A case of primary multiple endothelioma of bone. Arch. Surg., 1924, ix, 636. Idem. Angio-endothelioma of bone. Arch. Surg., 1926, xii, 854.

Fig. 76. Case 291. Skeletal angio-endothelioma. Showing proliferation and piling up of blood vessel endothelium.

The tumor cells are arranged in alveoli and tubules. The interalveolar septa form the base upon which the tumor cells are lined (Fig. 74). The cells may vary with the maturity of the tumor. In the young areas the alveoli are small, filled with tumor cells, without any perceptible lumina. The tumor cells are large polyhedral sometimes cylindrical with a well defined cell membrane and with pale vesicular nuclei and minute nucleoli. At a later stage the alveoli show lumina, sometimes filled with circulating blood. At a still later stage the tumor cells are undergoing degenerative changes. The most interesting feature is the frequent lining of alveoli with intact blood by a single row of tumor cells well simulating vascular endothelium (Fig. 75). It is not uncommon to be able to trace the origin of the tumor cells to normal appearing blood vessel endothelium (Fig. 76). In some cases the tumor cells do not enclose intact blood but form small alveoli or small cysts containing mucinous material. The cells may be more cylindrical and quite resemble carcinoma. A common feature is the fact that the strands of tumor are intimately connected with the supporting stroma and the surrounding tissue and are not separated from it by means of endothelial cells lining the connective tissue spaces, as is always seen in carcinomatous metastases. Well differentiated and low grade new formed bone may be seen

throughout the tumor. The question of the relation of the supporting stroma to the tumor elements is disputable; there is strong evidence on hand, however, that endothelioma may give rise to intercellular substance and fibrils as was described by Lubarsch and Marchand with reference to serous endothelioma.

There is described in the literature another type of endothelioma, a so-called intravascular variety which to my knowledge is not represented in the Registry material at all. This variety, usually multiple, consists of diffuse cords and sheets of clear polyhedral cells often surrounding capillaries with circulating blood. The possibility of the relationship of these tumors to myeloma is inadequately recognized, and the possibility is greater since these tumors have a myelogenous origin and not an endothelial one.

Clinical Course

Clinically angio-endothelioma of bone so much resembles osteogenic sarcoma that it is commonly submerged under the diagnosis of "bone sarcoma." The tumor is too rare a condition to allow definite conclusions as to the clinical incidence, age, and site of predilection. The fact that only two cases were recognized by me in the Registry material suggests that the clinical incidence is extremely low. The tumors may be encountered in old as well as in young. They seem to be prone to occur in any bone of the skeleton, especially in the long bones. Subjectively the clinical symptoms and signs are in general the same as those in osteogenic sarcoma, save perhaps for a later date of appearance of pain. The tumors may grow to a large size. In one of the cases (No. 154) the tumor led to absorption and destruction of bone while in the other (No. 291) a definite inclination to bone production was evident. In the first case no bone capsule was present and the periosteum was found free of any osseous tissue while in the second bulky osteoblastic tumors were produced. It is thought that angio-endotheliomata have a tendency to remain long enclosed in their investing capsule. Apparently the tumors may be single and multiple, while it is impossible to determine whether or not the multiple tumors are metastases of one primary tumor. The tumors may produce metastases through the blood and lymph stream, ending fatally from pulmonary metastases. While the clinical course is slightly slower than that of an average osteogenic sarcoma, the outcome is the same.

Diagnosis

The rarity of angio-endothelioma of bone and the scarcity of material available for study make obvious the peculiar diagnostic difficulties of this tumor. Unlike other malignant bone tumors the history and clinical appearance of angio-endothelioma is of no diagnostic significance. For the same reasons little can be expected from the radiological examination. Indeed, there are no points of diagnostic value because of the diversity of features of this tumor, the tumor

occurs in young and old, solitary and multiple, destructure—with apparent complete washing out of the bone shadow in the roentgenogram—and productive—with formation of large bulky bony growths. The diagnosis rests upon the histological structure of the tumor. But here also numerous pitfalls make a correct diagnosis difficult. The salient features of the histological structure of angio-endothelioma have been pointed out above. There are, however, certain points which are of especial importance in diagnosing the tumor. One of the main features which cannot be overemphasized is the fact that the presence of blood between acinous clusters of tumor cells is not to be considered as evidence that the cells formed a vascular space during the life of the tumor. Neither does the so-called perithelial arrangement of tumor cells mean that the tumor is an angio-endothelioma. The lack of realization of this fact has led to the erroneous diagnosis of these tumors as angio-endothelioma in numerous instances.

Another important and frequent source of error arises from too large expectations of a study of morphology of the tumor cell. There are no absolute signs of an endothelial cell, and one can never be certain from the morphological appearance that a given cell is really endothelial. From this fact, one infers that unless the origin of the tumor cells can be traced to vascular endothelium, the diagnosis of angio-endothelioma is not to be considered unquestionable.

In every case of suspected angio-endothelioma the possibility of a metastatic origin must be considered and eliminated. It is imperative to remember that a hasty pathological examination may lead to an erroneous diagnosis. In the literature cases of angioma and endothelioma of soft parts with secondary involvement of the adjoining bone were often confounded with angio-endothelioma.

Since angio-endothelioma does not respond in a noticeable degree to radiation, a therapeutic radiation test is probably of no promise. An exploratory incision is of doubtful advisability in these tumors; while such incisions are subject to all hazards of cutting into a malignant tumor the diagnostic advantages of exploration promise little here since no method is known to influence the course of the tumor or to prevent metastases after radical surgery.

Therapy and Prognosis

The question of the therapy in angio-endothelioma as it stands today is wide open. We have not the slightest conception about influencing the growth and fatal course of this tumor. The few cases observed do not allow any valuable conclusions as to the therapy. The limited experience gained, however, does show that there are no surgical cures and no improvement from radiation, roentgen ray, or radium; this despite the vascular structure of the tumor. Early metastases and multiple origin of these tumors still more aggravate the prognosis of this disease. Purely symptomatic relief is the only help that can be offered patients afflicted with this disease.

EXTRAPERIOSTEAL SARCOMA

Pathology

As was pointed out in the chapter on classification, by the term extraperiosteal sarcoma is understood a tumor which is intimately related to the periosteum but is entirely extracortical, not invading or infiltrating the bone. This tumor arises from the fibrous layer of the periosteum and not from the bone producing cambial layer. Thus, this tumor arises from nonspecific fibrous tissue which is incapable of producing osseous tissue. This difference between this tumor and osteogenic sarcoma substantiates the pathology, gross and microscopic, the radiological features, and the clinical course. Since this tumor originates from or is attached to the fibrous layer of the periosteum its investing capsule is not formed by the periosteum as is the case in osteogenic sarcoma. These tumors remain encapsulated for a long time and in their growth they push aside the soft tissues rarely infiltrating them.

While as a rule these tumors do not invade the bone, they may, however, cause absorption or slight erosion of the bone as a result of pressure, or they may push aside the adjacent bone and cause deformities. In cut section the tumor is firm, fibrous, white, glistening, with easily distinguishable fasciculi; occasionally it is cellular, soft, and crumbly, when regressive changes are extensive. In such tumors we see necrotic areas with formation of cysts filled with a dark brown fluid, probably a result of necrosis with extravasation. Histologically these tumors do not show any relationship to bone and this relationship is to be judged from the gross anatomy. The leading histological picture of this tumor is that of a fibrosarcoma. Its various sized cellular elements are of the spindle cell type. These cells resemble fusiform fibroblasts and when loosely arranged their granular cytoplasm can be easily seen. When compactly grouped, these cells show no cell borders, the nuclei seemingly occupying the whole field. Elongated processes of the cytoplasm form a distinct intercellular substance consisting of abundant fibrils. With the increase in size, the tumor cells may be pleomorphic. By special methods of staining one can readily bring out a fibrillar intercellular substance loosely arranged between borders of spindle cells. In rapidly growing tumors one sees abundant capillaries consisting of single rows of endothelial cells; on a superficial examination it appears that blood courses in lumina composed by tumor cells without any help of endothelial cells.

Clinical Course

The very few reported cases in the literature and the very limited number of tumors resembling extraperiosteal sarcoma on record in the Registry seem to indicate that the clinical incidence of this disease is very low. The difficulty of distinguishing with certainty these tumors histologically from fascial sarcoma and the ease with which the relationship of these tumors to the periosteum may remain unrecognized at operation probably cause a misrepresentation of the clinical incidence. In their onset, course, and physical findings these tumors hardly differ from fascial sarcoma. The relationship of the tumor to the bone, a broad attachment to the periosteum, may not be judged from the pre-operative findings on palpation; large tumors may be freely movable despite their attachment to the periosteum of the bone. There does not seem to be any definite relationship between the incidence of the tumor and age; I have observed one case of extraperiosteal sarcoma of the tibia in a newborn child, at present alive and well, 10 years after amputation of the thigh.

Diagnosis

With the difficulty of distinguishing an extraperiosteal sarcoma from a fascial sarcoma even at operation it is evident that the pre-operative diagnosis must be largely provisional. The roentgenogram, however, may give some points suggesting the diagnosis. Usually the roentgenogram shows an intact or slightly eroded shaft of the bone running through a tumor mass which is poorly outlined because of the faint shadow that it casts. The tumor does not surround the bone circularly. Since the periosteum does not actively react to the presence of the tumor one does not see here the periosteal spindle or lipping so frequently manifested in osteogenic sarcoma. Since the tumor does not invade the bone but may cause pressure deformity one may encounter roentgenologically changes in the contour of the bone. Occasionally the tumor may lead to proliferative processes of the bone to which it is attached; the outline of the bone then remains smooth as in benign osteogenic tumors. Sometimes the tumor causes superficial erosion of the bone. In such tumors of fingers or toes one is reminded of gout when the bone is eroded, but instead of being rarefied, atrophic, it is denser than normal. A gout tuft can be easily recognized grossly from the gritting of the uric acid crystals when cut with a knife (Plate 8).

Therapy and Prognosis

Radical surgical removal is the operation of choice in extraperiosteal sarcoma. Thorough incision may be followed by permanent recovery. A considerable number of cases reported in the literature as cures of "bone sarcoma" after surgical operations probably belong to the extraperiosteal sarcomata. The

prognosis is especially favorable when the tumor is found encapsulated. After incomplete excision these tumors may recur but with a moderate tendency to produce metastases. Recurrence is not unusual in the large, more actively growing tumors in which regressive changes are frequent. With the recurrence, the texture of the tumor may become more anaplastic and malignant. Not uncommonly the intimate relationship of the tumor to the periosteum is overlooked at operation, the extraperiosteal sarcoma is mistaken for a fascial sarcoma and the attachment to the periosteum is left behind. Surgical removal of a fibrous sarcoma located close to a bone therefore should be followed by prophylactic radiation therapy, preferably with roentgen rays.

GIANT CELL TUMOR

Pathological Concept and Etiology

THE clinical and pathological aspects of giant cell tumor of the skeleton are so at variance with the malignant skeletal tumors that a discussion of the disease here calls for explanation. As is inferred by the name, by giant cell sarcomata are understood lesions the histological structure of which is characterized by an abundance of giant cells. However, such a definition of giant cell sarcomata would be erroneous since not all tumors showing the presence of giant cells belong here. Three types of giant cells can be distinguished in pathological conditions. First come the giant cells which are a result of karyorrhexis in the cellular elements of the tumor. This is especially frequently observed in rapidly growing malignant tumors where the division of the cytoplasm cannot keep pace with the closely following, repeated divisions of the nucleus. The nuclei here are of various sizes and shapes, frequently not completely separated from each other. These giant cells are a significant part of the parenchyma of the tumor. Following the lead of Mallory these are spoken of as true tumor giant cells. Another type is the so-called foreign body giant cell. These occur in tuberculosis and in gummata; they are the Langhans type of giant cells with a peripheric or bipolar arrangement of their nuclei. The third type is represented by the giant cells of the so-called giant cell sarcoma which is here designated as giant cell tumor. The nuclei are uniformly distributed throughout the central portion of the cell; they are of equal size and completely separated from one another. The fact is still insufficiently realized that only lesions with giant cells of the third type are understood under the term of giant cell tumors. Skeletal lesions designated as giant cell sarcomata are of three varieties: (a) the giant cells containing new growths observed in the course of osteitis fibrosa, (b) the epulis, and (c) the giant cell tumor. The lesions marked under (a) are generally conceded to be of purely inflammatory character and obviously do not belong in the present analysis. Epulis, as the term infers, is a lesion of the gums or alveolar periodontium rather than of the bone itself. The benign course of epulis has been generally realized for a long time and to include epulis in our present study of giant cell tumors could be justified merely for reasons of emphasis, for such an association would tend to stress the point of benignity of giant cell tumors.

The term giant cell tumor is applied to a condition which has been and still is known in various countries and even in one and the same country by different

names. *Tumeur à myéloplaxes* given by Nélaton has changed to myeloid sarcoma and myeloma, which is still being used in the British literature; it is also known as giant cell sarcoma, benign giant cell sarcoma of the epulis type, giant cell sarcoid, osteitis fibrosa cystica, hæmorrhagic osteomyelitis, and giant cell tumor. The benign character of giant cell tumors is not yet generally recognized, and not infrequently the lesion is still looked on as a sarcoma. The fact that hundreds of cases of giant cell tumor have been sent to the Registry of Bone Sarcoma by clinicians and pathologists the country over is sufficient justification of the necessity of the discussion of this lesion in a monograph primarily dedicated to malignant bone tumors.

The question of the etiology of giant cell tumors is at first glance intricately interwoven with an academic question: Is giant cell tumor a neoplasm or a product of inflammation and repair? The acceptance of the name giant cell tumor as a substitute for all other names of this lesion is not to be looked upon as proof of the neoplastic nature of the lesion; the term "tumor" is used here merely in a clinical sense, that is, to indicate a swelling, inasmuch as the pathological anatomical sense of this word is at present not entirely clarified. The question of whether a giant cell tumor is a true blastoma or merely an inflammatory process follows an old trodden path of discussion; a similar question was in vogue twenty-five years ago in relation to the new growths observed in the course of osteitis fibrosa. It was Mikulicz who then in the light of contemporary knowledge warned against the wrong assumption that bone cysts are results of true tumors which have undergone softening and degeneration. As it stands today the concept of the disease known as giant cell tumor divides investigators in two opposing classes. To one belong those who look upon a giant cell tumor as a true blastoma and to the other those who see in it merely a product of inflammation and repair in bone. Since the view upon giant cell tumor as a true blastoma is supported by tradition, the burden of proof in this dispute lies on the promulgators of the inflammatory nature of giant cell tumors. The leaders among these are Mallory, Codman, and Barrie in this country and Lubarsch and Konjetzny in Europe. Mallory has long maintained that the giant cells of the giant cell tumor are not an integral part of the lesion but only a biological reaction of the large mononuclears of the blood, the so-called endothelial leucocytes, which are found wherever retrograde changes are going on. As a reaction to calcium salts absorption these endothelial leucocytes fuse and form the giant cells. Aside from these giant cells no cells occur in these lesions which are not met with in ordinary inflammatory processes. Of the same opinion is Codman who sees in the giant cell tumor a repair process following intra-osseous hæmorrhages due to rupture of nutrient vessels. In Codman's opinion the tendency of this disease to form large expansive tumors does not warrant considering it as a neoplasm any more than does the enlargement of an aneurysm.

Strongly substantiated by results of special investigations are the opinions of Lubarsch and Konjetzny. Years ago Lubarsch had pointed out with the clarity and logic usual for him that the new-growths observed in the course of osteitis fibrosa are of purely inflammatory nature. Later he joined Konjetzny in the latter's interpretation of results obtained from special studies of giant cell tumors. On the side of histological preparations Konjetzny showed how an intermedullary hæmorrhage calls forth a reactive proliferative process. The product of this proliferative process of the bone-marrow which can be compared to granulation tissue consists histologically of all the elements encountered in lesions known as giant cell tumor. The clinical course and the radiological findings are very closely related to those of giant cell tumors. In the course of the natural life of this granulation tissue there is a stage of differentiation when fibrous tissue, osteoid, and sometimes osseous tissue take the place of the hæmorrhage after it has subsided, the blood clot organized, and all the foreign elements removed. Thus Konjetzny concludes that the apparent tumor is merely a "chronic-resorptive process."

The opponents of the opinion that giant cell tumors are true blastomata emphasize the following points of the histology of giant cell tumors as supporting their views: the absence of pleomorphism of the cellular elements and of hyperchromatism of the nuclei and the absence of excess of mitoses; the differentiation of the cellular stroma into dense fibrous tissue poor in cells; the uniformity in the size, shape, and chromatin content of the giant cell nuclei; the relation of the giant cells to extravasations, indicating their rôle in resorption; and the constant presence of old blood pigment. All these features are not inconsistent with the probable inflammatory nature of these lesions. If one admits the correctness of the views of these authors with reference to certain skeletal lesions and especially to the new growths occasionally observed in the course of osteitis fibrosa one is, however, prompted to accept the contention that along with these lesions there probably are also true blastomata very similar to these clinically and histologically. It is generally conceded how difficult it is to distinguish histologically between a new-growth in osteitis fibrosa and entirely independent lesions considered as giant cell tumors. Of course this fact alone is sufficient evidence to raise doubt as to the right by which giant cell tumor is occupying its place in oncology. On the other hand there is no sufficient evidence accumulated to support the contention that in all cases of giant cell tumor the lesion is a process of inflammation and repair.

The etiology of the typical giant cell tumor is readily understood if one accepts the new-growths observed in the course of osteitis fibrosa cystica as giant cell tumors. The term "osteitis fibrosa" is being greatly misused of late. The average roentgenologist is usually inclined to call osteitis fibrosa most of the bone lesions which he is unable to diagnose and classify. This fact well illus-

trates the need of further study of this little understood pathological condition. The main complex of osteitis fibrosa is the disappearance of hæmatoblasts and fat cells from the bone-marrow with a subsequent overgrowth of the fibrous stroma and lymphoid elements. This is accompanied by a simultaneous resorption of bone and new formation of osteoid tissue, and this leads later in the course of years to fractures and deformities. Frequently, in the course of the disease one encounters formation of cysts. Hæmorrhage into the cysts may lead to an overgrowth of masses, resembling granulation tissue, which may enlarge and finally be recognized as giant cell tumors. The purely inflammatory nature of these lesions has been emphasized above. The frequent history of an antecedent mild trauma in giant cell tumor has led to the recognition of trauma as an important etiological factor. As in malignant bone tumors, much depends upon the individual concept of trauma. It has been shown that a hæmorrhage into the bone-marrow may lead to cyst formation as it is observed in the brain. Organization of the intramedullary hæmatoma with the appearance of the peculiar medullary granulation tissue and expansion of the bone will then form the giant cell tumor. Very slight traumatization is frequently sufficient for the appearance of medullary hæmatomata. While it is usually thought that fractures are not the cause but merely a complication of giant cell tumor and osteitis fibrosa, it is probable that fissure fractures or, better, infractions with the rupture of nutrient vessels may lead to the formation of a giant cell tumor. The frequent presence of old hæmorrhage histologically in a giant cell tumor and the almost exclusive location of giant cell tumor in the ends of the bone in the spongiosa seem to support this view. Wright and Ewing brought forth evidence of the origin of variants of giant cell tumor from absorption of aberrant islands of cartilage cells which are not unusual in patients suffering from rickets in childhood. This absorption may lead to a neoplastic proliferation of the released cells. All mentioned factors, however, fall short of a satisfactory explanation of the etiology of all varieties of giant cell tumors and cases are encountered in which the etiological factors remain obscure for the present.

Pathology—*Gross Anatomy*

As in osteogenic sarcomata, the gross anatomy of giant cell tumors depends largely upon the destructive and productive processes of the involved bone. The tumor tissue during its period of growth constantly destroys the bone while the periosteum lays down an advancing shell of new bone, thus preventing the tumor mass from an early perforation of the bone and an involvement of the adjoining structures. It is due to a combination of these two opposing reactions of the involved bone that the giant cell tumor is an expansible but not infiltrative or invasive lesion encapsulated in a bone shell. Eating its way into the bone, the tumor tissue destroys the bone from within the medullary cavity, gradually

Fig. 77. Courtesy of Warren Museum, Boston. A macerated specimen showing the skeleton of a giant cell tumor. It shows the sharp limitation and general expansile character of the tumor. Notice the extension over the top of the fibula without involvement of the latter. In some cases the tumor enters the fibula by way of spreading the ligamentous attachments between the bones (Plate 35). A partial pathological fracture is seen.

expanding the old cortex, while a few more or less thick bony trabeculæ running in various directions line the cystically expanded bone shell simulating beams supporting the whole structure. It is this structure that causes the most characteristic "soap bubble" appearance in the roentgenogram. The structure of a skeleton of a giant cell tumor is best studied in the macerated and dried specimen when the soft spongy tissue is removed (Fig. 77). Thus the investing capsule of the giant cell tumor is furnished by the new formed bone shell and the periosteum. The latter, while distinctly thicker and more vascular than on the adjoining uninvolved cortex, does not form, however, the abrupt elevation, the periosteal lipping, which is so typical of osteogenic sarcoma. When nothing interferes with the advancing growth of the tumor, the bone shell may be gradually thinned and finally entirely disappear in places. Long after the bone shell is thus perforated the periosteum still continues to envelop the tumor until a very advanced development of the tumor or a pathological fracture hastens the perforation of the periosteal capsule. This is in marked contrast to a malignant bone tumor where the perforation of the bone and periosteum occurs very early. Another factor leading to perforation of the periosteum in giant cell tumor is an exploratory incision although followed by suture of the periosteum. On a longitudinal section through a giant cell tumor

Fig. 78. Case 454. Same case as that in Figure 84. Giant cell tumor in a man 28 years old. The arrow points to the limiting membrane of the lesion, toward the medullary cavity.

one occasionally recognizes grossly a limiting membrane toward the medullary cavity; a thin layer of brown whitish tissue beyond which the normal spongiosa or bone-marrow can be seen (Fig. 78).

The articular cartilage is very resistant in giant cell tumor as it is in osteogenic sarcoma. It is most unusual to see the articular cartilage destroyed by tumor tissue even in the advanced stage. Usually the spongiosa lining the articular cartilage from within the medullary cavity is entirely destroyed and the cartilage is in immediate contact with the tumor tissue (Plate 34). In such a proximity of the tumor to the joint cavity one may find the synovial membrane slightly thickened, œdematous, of brownish yellow color. It is not unusual to find some clear exudate in the joint cavity. An actual direct involvement of the joint cavity by tumor tissue is even less frequent than in osteogenic sarcoma; it occurs in pathological fractures and in very advanced cases. It is not unusual, however, to see an indirect involvement of the joint cavity by tumor spreading along intra-articular ligaments, for instance along the ligamentum teres femoris or ligamenta cruciata. The same manner of growth of the tumor, by spreading the lamellæ of ligaments, one sees also in involvement of an adjoining bone when the ligamentous attachments between the bones usually serve as a bridge for the

Plate 34. Case 580. Giant cell tumor. The roentgenogram was taken before amputation. The extent of involvement in giant cell tumor is always greater than it appears from the roentgenogram. The apparent limitation of the tumor to the external condyle is misleading as is seen from the photographs. Above, right, section through the external condyle; it shows the intact articular cartilage, while the entire condyle is displaced by the lesion. Below, right, section through the internal condyle.

tumor (Plate 35). This way of expansion of the tumor along lines of lesser resistance is also observed in the process of perforation of the periosteal investing capsule where the layers of the periosteum are spread by the ingrowing tumor tissue; it is also seen in the spreading along fascial planes of the tumor mass projecting into the soft tissue after periosteal perforation. In far advanced and large giant cell tumors the expansion of the bone shell is interfered with by the surrounding tendons and ligaments, so that occasionally the roentgenogram, but more frequently the dried specimen, shows grooves in the bone shell through which the individual tendons ran (Fig. 79).

The gross appearance of the giant cell tumor depends greatly upon the phase of the lesion. The typical giant cell tumor consists of solid portions and numerous small cysts. The solid portions are very friable, crumbly, somewhat granular masses, varying in color from yellow and light brown to dark red. On incision the tumor tends to extrude like granulation tissue. The texture of the growth becomes more dense with the approximation to the periphery and capsule. This vascular, soft, readily oozing, and frequently profusely bleeding tumor, resembling currant jelly, is entirely confined within the bone shell; it lies there loosely and can be easily scooped out by a curette. With the aging of the tumor or after radiation therapy the tumor mass enters a cicatrizing phase. The reddish jelly-like tumor mass changes gradually, beginning at the periphery, to a more opaque and firm mass, while in the central portion the old juicy stroma prevails. With the advancing differentiation of the tumor tissue, fibrosis in all stages to fibrosis and cicatricial tissue may be seen. Some varieties of giant cell tumors may be solid and firm throughout from the commencement and because of their frequent peculiar yellow color due to the presence of lipoid material they have been designated as xanthomata. The variety of giant cell tumors which, according to Wright and Ewing originates from absorbing of aberrant cartilaginous islands, shows the presence of various sized foci of cartilage distributed in the giant cell tumor mass. The various phases of giant cell tumor are a result of advanced differentiation of the soft immature tumor tissue. All successive processes of repair can be traced here. Such a differentiation is especially marked in well advanced cases. A pathological fracture through the tumor or radiation therapy, as well as an incomplete surgical curettage, may give rise to a cicatrization of the tumor. When a giant cell tumor is complicated by a fracture the soft hæmorrhagic giant cell tumor tissue is replaced by active fibrosis which is followed by peripheral ossification. Usually, however, the outer part of the tumor alone is fibrosed, the central portion remaining in its immature stage or being replaced by a fluid containing cyst. The firm, dry, fibrosed peripheral portion may show osteoid tissue formation and even ossification.

At the suggestion of Ewing an attempt has been made to differentiate several varieties of giant cell tumors which differ from the typical tumor grossly as well

Plate 35. Case 212. Giant cell tumor in a man 24 years old. Curettage on several occasions led to a persistent sinus with tumor sprouting through it. The roentgenogram was taken before the amputation, 2 years and 2 months after the onset. The roentgenogram and especially the photograph show the spreading of the tumor along the tibia—fibular attachment into the fibula.

as histologically. Thus, as xanthoma is designated a variant of giant cell tumor in which the presence of considerable fatty detritus lends the tumor a yellow appearance. Another variant is the so-called myxomatous giant cell tumor in which a mucinous matrix is the main characteristic feature. Giant cell tumors in whose texture islands of cartilage are encountered are said to arise from absorption of misplaced islands of cartilage, when the released cartilage cells acquire a neoplastic character. In this variety of giant cell tumor it is not unusual to see extensive areas of calcification about the remnants of cartilage cells. As in osteogenic sarcoma here also the degree of vascularity of the tumor has been laid as a basis of differentiation of a so-called telangiectatic giant cell tumor. Already in a typical giant cell tumor the central portion is made up either of an organizing blood clot or a sinusoidal reticulo-endothelial system filled with grossly intact blood, reminding one of a bloody sponge. When the extensive vascularity is confined not merely to the center of the tumor but is present in the largest portion of the tumor, it then represents the telangiectatic variety of giant cell tumor

(Plate 36). The clinical importance of such a differentiation of variants of giant cell tumor is questionable since there is no sufficient evidence accumulated to support the contention that these variants differ greatly in their clinical course. Furthermore, such variants cannot be distinguished before the tumor is submitted for a pathological examination, gross or histological.

The rich vascularity of giant cell tumor alone suggests the great tendency of the tumor tissue to retrograde change; the frequent presence of blood clots, of old blood pigment, and of areas of hæmorrhage in all stages of decomposition is sufficient evidence of the frequency with which regressive changes occur here. After radiation therapy of a giant cell tumor it is not unusual to see in the central portion extensive necrosis and cyst formation filled with blood clot and serous fluid. The occurrence of cysts in an advanced giant cell tumor is not uncommon. Occasionally the polycystic appearance is very striking. Various cysts, reaching the size of a hazel nut, are confined within chambers formed by brownish septa, at times paper thin and then again of considerable thickness, interwoven among themselves and at the periphery rather firmly attached to the bone trabeculæ of the bone shell. The cysts are filled with blood or chocolate colored fluid (Fig. 80); or an opaque, brownish, or greenish mucinous mass, a greenish or grayish serum; evidently the product of various stages of decomposition of blood extravasations.

Fig. 79. Courtesy of Warren Museum, Boston. A dried specimen of a giant cell tumor of the lower end of the radius. Showing the grooves for the tendons and the ballooning of the styloid process. The nodule at the top of the styloid process apparently belongs to the carpal bones. The patient, a woman, died in 1850 from a natural cause.

Structure

The histological structure of the typical giant cell tumor consists of two leading elements: the giant cells and the stroma. The latter consists of numerous

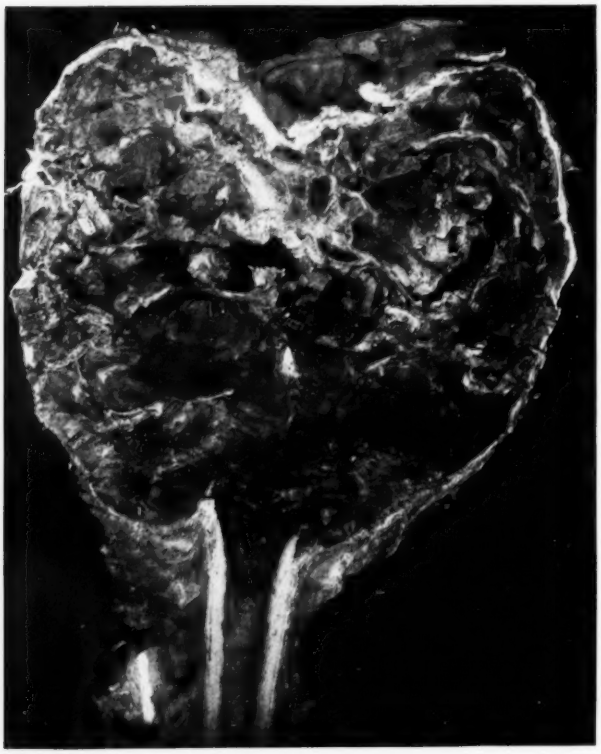


Fig. 80. Case 320. Same case as that in Plate 36. Giant cell tumor of the tibia in a woman 35 years old. Amputation 3 years after the onset. Showing complete absence of a periosteal reaction on the adjoining portion of the uninvolved shaft.

various sized blood spaces and exceedingly thin walled capillaries suspended in a very loosely woven net of spindle, round, or polygonal cells with large vesicular nuclei. One of the most characteristic features of a giant cell tumor is the absence of pleomorphism of the cells of the stroma. This loose cellular tumor mass is very poor in fibrils in contrast with the peripheral portion of the tumor where the differentiation of the cellular elements usually begins first, and where the stroma is poorer in cells but firmer through the abundance of fibrils. In the central portion of the tumor fresh extravasations of blood as well as hæmosiderin can be found; the latter, however, is usually found near the periphery of the bone shell. With the advancing growth of the tumor the blood clot may organize, and the blood pigment is carried away by giant cells and by the abundant endothelial leucocytes sprinkled through the tumor mass. It is in these endothelial leucocytes and not in the cells of the stroma that mitoses are not infrequently observed.

The numerous giant cells are loosely embedded in the stroma (Fig. 81). These are large opaque acidophile cells, resembling normal osteoclasts and containing numerous small oval nuclei uniformly shaped with a chromatin content larger than the nuclei of the stroma cells. The nuclei are grouped in the central portion of the large cells. The histogenesis of this peculiar cell is still a subject of discussion; the theory in vogue at present is that these cells originate from the endothe-

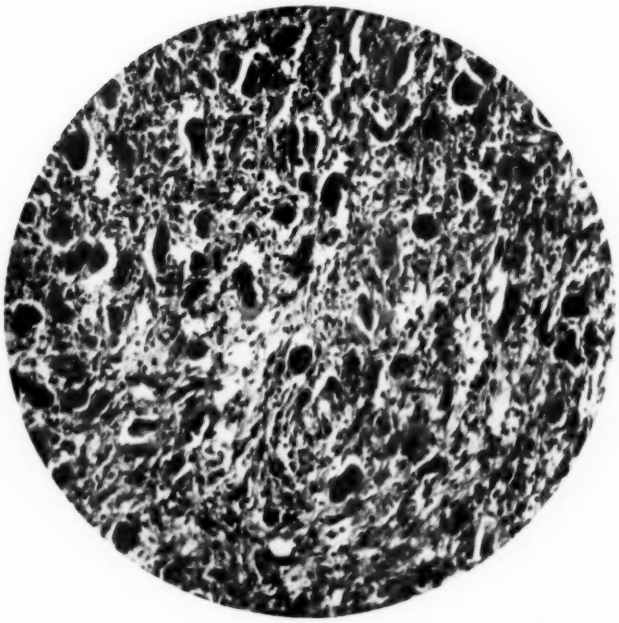


Fig. 81. Case 208. A typical structure of a giant cell tumor. Abundant giant cells of epulis type are loosely embedded in a fibroblastic stroma.

lium. The very intimate relation of the giant cells to the vascular endothelium, the frequent lining of blood spaces by giant cells, and the tracing of the formation of giant cells to the proliferative vascular endothelium seem to support Lubarsch's view of the giant cells as abortive vascular sprouts. Mallory believes the giant cell to be a product of fusion of endothelial leucocytes in the presence of calcium salts absorption. In a study of the giant cells as they are seen in various stages of giant cell tumors one can trace their composition from smaller cells as well as their splitting up in cells with single nuclei. Apparently after the need for the giant cell has passed they have a tendency to disunite. Frequently, however, one sees the retrograde changes leading to the terminal end—death of the giant cell. Minute clefts and small vacuoli appear in the cytoplasm; the latter are probably traces of fatty detritus which was phagocytized by the giant cell and removed from the cell in the course of the histological preparation. If the cytoplasm has not disappeared in the process of splitting up of the individual nuclei, it may remain as a dead, faintly stained clump of cytoplasm riddled with vacuoli and shadows of the nuclei. Such cytoplasm may break up in small débris and be finally removed from the tumor by other phagocytes (Plate 37, frontispiece).

Plate 36. Case 320. Same case as that in Figure 80. Giant cell tumor in a woman 35 years old. Radiation was attempted but unsuccessfully; apparently because of the immense size of the tumor. Amputation 3 years after the onset. Showing the huge blood spaces in relation to the main blood vessels.

Fig. 82. Case 142. Giant cell tumor. Showing the net-like structure of the spongy tumor with the giant cells playing an active rôle in the texture of the tumor.

There is at present much discussion whether the giant cell is an integral part of the tumor or merely a side product called forth for the scavenger work in the lesion loaded with decomposing material. Whatever opinion one may hold, one cannot deny that the structure of a typical giant cell tumor with the abundant giant cells tied up like knots at the junctions of endothelial strands suggests that the giant cells play an important rôle in the composition of the tumor (Fig. 82). It is true that, when needed, the giant cells take up the function of scavengers and it is not uncommon to find in giant cells fatty detritus, remnants of blood cells, blood pigment, and even small spicules of bone.

Apart from giant cells one may occasionally encounter in the stroma of a giant cell tumor a lymphocytic and myelocytic infiltration which is not unusual for a medullary lesion. In tumors of long standing one sees an advanced differentiation of the fibroblastic stroma entering a cicatrizing and fibrosing stage. Such a differentiation may be hastened by radiation therapy or by a spontaneous fracture through the tumor. Usually the differentiation proceeds from the periphery to the center of the tumor but occasionally one encounters a differentiation in islands in the middle portion of the tumor while the more peripheric portions

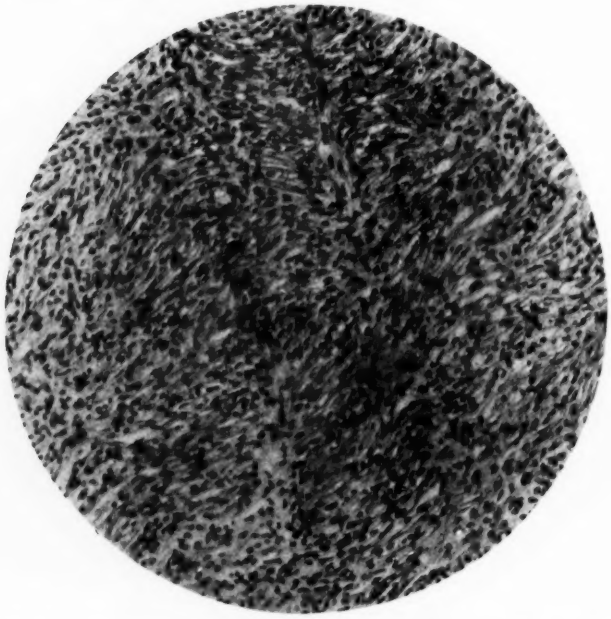


Fig. 83. Case 103. Giant cell tumor; cicatrizing and fibrosing stage. The stroma is represented by a dense fibrous tissue poor in giant cells.

temporarily remain immature. The spindle fibroblasts become smaller; fibrils appear about them, and soon the stroma is represented by a dense fibrous tissue poor in cells (Fig. 83). Osteoid tissue formation and metaplastic ossification can be expected here.

To avoid errors in diagnosing a giant cell tumor from the histology one has to keep in mind the various deviations from the typical giant cell tumor structure. Probably the most frequent variation of giant cell tumors is the so-called xanthoma, when the resorption of a considerable amount of fatty detritus leads to an impregnation of the phagocytozing cellular elements with lipoids. Typical giant cells are few there, and their place is taken by aggregates of endothelial leucocytes which are peculiar here because of the fairly granular cytoplasm resulting from the lipoid inclusions. These are the so-called "foam cells" (Fig. 84). A deviation from the typical giant cell tumor is also observed in the myxomatous variation, the peripheral portion of which consists of spindle cells, slightly smaller in size with a large chromatin content of the nuclei, embedded in a mucinous mass (Fig. 85). The central portion may retain all characteristic features of the typical giant cell tumor and even show areas of spontaneous healing with hyalinization and calcification (Figs. 86, 87).

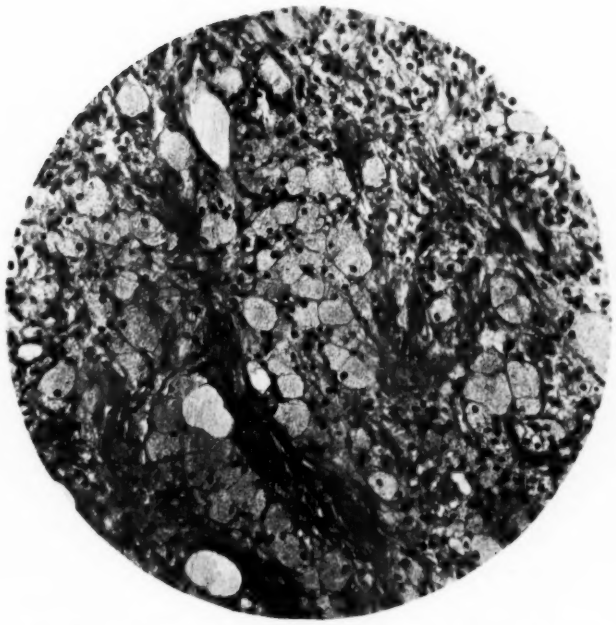


Fig. 84. Case 454. Compare with Figure 78. Giant cell tumor; xanthoma variety. Showing the "foam cells" filled with lipoid inclusions.

The variety of giant cell tumor originating in connection with an absorption of misplaced islands of cartilage, presents a marked deviation from the normal structure. Along with areas of degenerating cartilage undergoing absorption and calcification newly formed, imperfect cartilage cells can be seen as polyhedral granular cells arranged in sheets and clusters. At a glance they somewhat resemble epithelial cells and the name "epithelioid cells" has been suggested for them (Fig. 88). The degenerating cartilage is hyaline or dark staining fibrillar material. The spindle fibroblastic structure is absent here and a very cellular stroma composed of rounded cells gives the tumor the general aspect of a myeloma (Fig. 89). There are a few giant cells scattered in the cellular areas, especially about extravasations and blood spaces.

Unlike malignant bone tumors, in which the destruction of the involved bone is accomplished by both osteoclasts and tumor cells, in giant cell tumors the task of destruction of the cortex is taken over by the giant cells. The destruction of the bone shell by the giant cells from within and the apposition of new bone by a dense row of osteoblasts beneath the periosteum from without can be easily seen in section (Fig. 90). In the areas where the struggle between the giant cells

Fig. 85. Case 592. Same case as that in Figures 86 and 87. Giant cell tumor. This section was taken from the periphery of the tumor. Showing the myxomatous structure of the tumor.

and the osteoblasts resulted in a complete destruction of the bony capsule, the giant cells retreat from the advance guard of the tumor and the number of giant cells along the periosteal investing capsule is smaller than when remnants of bone were lining it (Fig. 91). The periosteum remains intact for a long time, forming the last wall of defense before the tumor breaks through into the surrounding soft tissues. Nothing contributes more to the hastening of such a perforation than an exploratory incision or incomplete curettage. Once incised the periosteal capsule remains open and disabled in its effort to encapsulate the tumor.

When a recurrence has taken place in a giant cell tumor the histology shows a variable picture and is always of an altered character. Cicatrization of a giant cell tumor may be hastened occasionally by incomplete curettage and sometimes even by an exploratory incision, provided infection does not set in. Frequently, however, infection is not avoided and the infected fungating pulsating masses of the tumor acquire an appearance of a malignant new-growth. Histologically such a giant cell tumor is greatly changed by the admixture of a reaction to infection and by stimulation to active growth.

Fig. 86. Case 592. Compare with Figures 85 and 87. This section was taken from the central portion of the tumor. Typical structure of giant cell tumor.

Clinical Course

The clinical incidence of giant cell tumors is apparently lower than that of primary malignant bone tumors. If one judges from the material of the Registry, the relative frequency of giant cell tumors as compared with malignant bone tumors is about 1:2. This ratio is probably an exaggeration of the frequency of giant cell tumors, since the Registry material counts many cases in which the patients were alive at the time the Registry began, while the average life duration of a patient with a malignant bone tumor is about 20 months. The giant cell tumor is more frequently met with in the female than in the male; the ratio 6 to 5 is probably a fair expression of this frequency The age in which giant cell tumor is most frequently encountered is expressed by the adjoining histogram (Fig. 92). It is in the decade between 16 and 25 that most giant cell tumors occur, an age considerably higher than for osteogenic sarcoma and Ewing's sarcoma. In 28 per cent of females the disease occurred after the age of 30 as against 41 per cent in males. The youngest patient was a girl of 6 and the oldest a man of 68.

The location of the giant cell tumor is in marked contrast to that of osteogenic sarcoma as far as the bone and the site of involvement is concerned. The bones of

Fig. 87. Case 592. Compare with Figures 85 and 86. Showing areas of spontaneous healing with hyalinization and calcification.

the lower extremity were involved in 56 per cent of all cases of the Registry while those of the upper extremity were involved in 23 per cent of all cases; in 21 per cent of all cases the bones of the trunk including the pelvis and shoulder girdle and the jaws were involved. Of all the cases of involvement of the upper extremity the radius was involved in 40 per cent, all in the lower end of the bone. The femur was involved in 57 per cent of all cases of giant cell tumor of the lower extremity and the tibia in 36 per cent. The lower end of the femur was, as a rule, involved, with very few exceptions, when the tumor was situated in the upper third of the femur about the trochanters. The lower end of the femur is involved much more frequently in the male than in the female while an involvement of the upper end of the tibia, which is three times as frequent as that of the lower end of this bone, is seen more frequently in women than in men. In general in about 47 per cent giant cell tumors were situated in the lower end of the femur and the upper end of the tibia. The jaws were the seat of the tumor in about 9 per cent of all cases, the spine following closely with 8 per cent of all cases of involvement. Involvement of the jaws which is equally distributed between the upper and lower jaw, is apparently rare after the age of 25. The shaft of the long bones was involved in two cases, while in both these cases there was place for doubt as to the

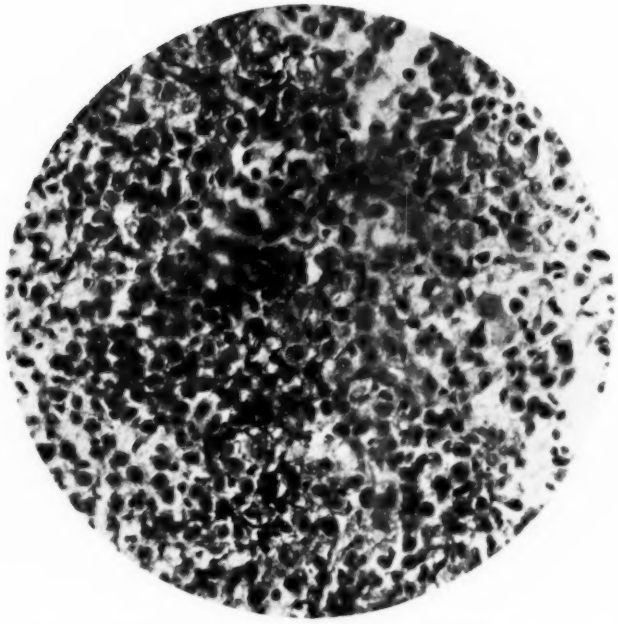


Fig. 88. Case 5. Same case as that in Figures 89 and 98. Giant cell tumor. Showing the "epithelioid cells."

accuracy of the diagnosis since a cyst complicated by fracture could not be ruled out. Giant cell tumor is situated in the small bones of the extremities more frequently than osteogenic sarcoma. In contrast with osteogenic sarcoma, in which the epiphysis frequently escapes involvement because of the epiphyseal cartilage serving as a barrier to the spreading tumor, in giant cell tumor the epiphysis is involved in the large majority of cases. Here the epiphyseal cartilage does not seem to exert any influence upon the spreading of the tumor and the latter frequently extends from here into the diaphysis. When the tumor is situated in the lower end of the femur or the upper end of the tibia, frequently one condyle or one tuberosity is involved; the tumor may spread into the diaphysis leaving the opposite condyle or tuberosity uninvolved. As a rule the giant cell tumor appears as a solitary lesion and it would seem probable that in some of the cases of multiple giant cell tumors one is dealing with a proliferative osteitis fibrosa.

The importance of trauma as an etiological factor in giant cell tumor has been mentioned above. If one is careful in gathering the facts of the patient's history, one very frequently encounters an antecedent local injury. In giant cell tumor of the jaw it is not uncommon that after a loose tooth is pulled a giant cell tumor develops; in such cases the injury apparently plays a stimulating rôle in the growth

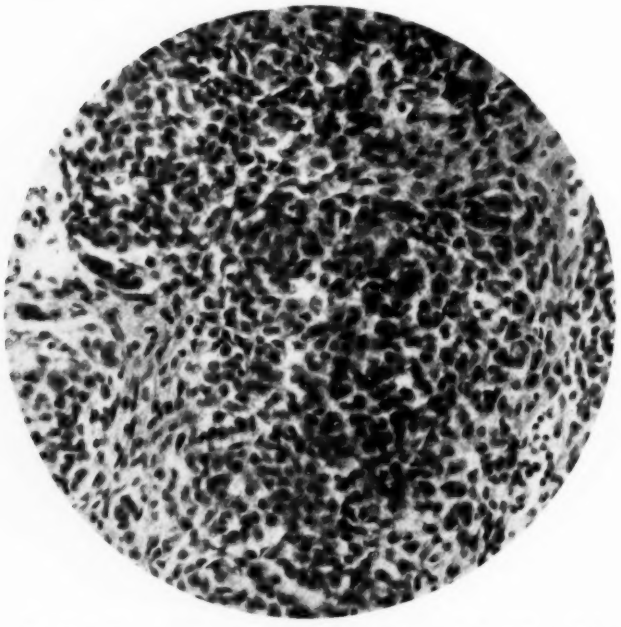


Fig. 89. Case 5. Compare with Figures 88 and 98. The cellular stroma consisting of round cells reminds one of myeloma.

of the tumor. Pain is frequent and early complained of by a patient afflicted with giant cell tumor. The pain, however, is of less severity than in osteogenic sarcoma, and it is more persistent after radiation therapy is begun. The patient's general condition usually remains good unless an exploration or incomplete curettage was done accompanied by infection. Infection is very persistent in giant cell tumor and it may lead to sepsis in a brief period of time. The skin frequently lacks the dilated veins commonly seen in osteogenic sarcoma. When the skin is very distended by the large tumor, it may resemble pig skin, be œdematous and cyanotic. Ulceration of the skin occurs apparently only in the very far advanced cases which have long gone without medical attention.

Palpation is an important diagnostic aid in the examination of a giant cell tumor. Frequently one is able to palpate the bony capsule of the tumor and its borders at the junction with the uninvolved diaphysis. One feels the bulky spheric shape of the tumor as contrasted with the fusiform shape of the osteogenic sarcoma. It is much easier to palpate in giant cell tumor because of relative absence of tenderness. When the bone shell of the tumor becomes very thin, egg-shell crackling can be made out. In the very vascular variety of giant cell tumors one occasionally feels a bruit. As a rule giant cell tumor is of long duration

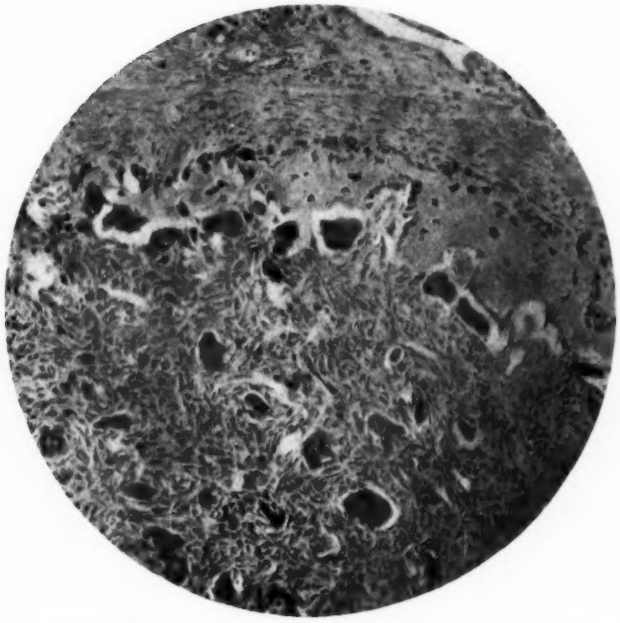


Fig. 90. Case 235. Same case as that in Figure 91. Giant cell tumor. Showing the destruction of the bone shell by the giant cells from within and the apposition of new bone by rows of osteoblasts beneath the periosteum.

and slow growth; notable exceptions are known however. At the present day advanced stages of giant cell tumors are seldom seen since their growth is interrupted by surgery or radiation. Occasionally in long standing tumors attempts at spontaneous healing occur,—cicatrization with ossification of the peripheric portion and cyst formation in the center. When infection takes place fatal hæmorrhage and sepsis may ensue. After breaking through the investing capsule the tumor travels along the intermuscular and fascial planes but does not invade the muscle tissue. In giant cell tumors of the spine a compression myelitis may offer an orthopedic issue. Frequently such patients die from supposed sarcoma while occasionally they can be saved by orthopedic measures. Infraction is almost a rule in giant cell tumor, especially in the weight bearing bones, where also complete pathological fractures are frequent. This fact again emphasizes the necessity of splinting and recumbency. The pathological fracture of the lower extremity is usually of the telescoping variety, with one end of the bone projecting into the cystically dilated other end. Joint involvement is exceedingly rare. When it occurs it frequently is due to undermining of the articular cartilage, which process is followed by collapse of the joint and rupture of the tumor into it.

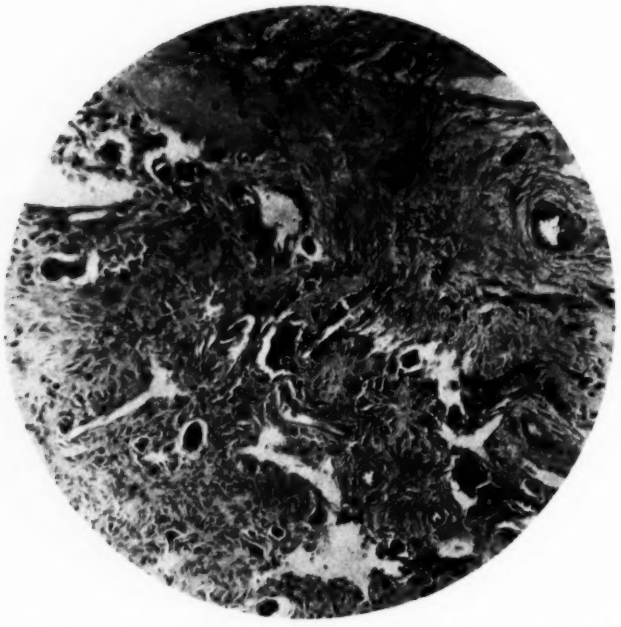


Fig. 91. Case 235. Compare with Figure 90. When the bone shell is destroyed the giant cells retreat from the advance guard of the tumor, and the number of giant cells along the periosteal investing capsule is scarce.

Diagnosis

Most of the general points in diagnosis outlined in the chapter on osteogenic sarcoma are applicable also in relation to giant cell tumors. While some experienced observers maintain that it is always possible to arrive at a diagnosis in giant cell tumor from a microscopic examination of the section alone, and others claim that the clinical history together with the physical findings and roentgen-ray features will suffice for diagnosis without an exploration of the tumor, there are times when neither method of examination, clinical and radiological, or microscopic, nor both of them combined will suffice for an accurate diagnosis. These cases are rare, frequently they are complicated by previous surgical treatment. These are the cases referred to by Ewing as the "borderline" giant cell tumors, with a wide destruction of bone, with an absence of the bone shell with smaller than usual giant cells containing larger and more hyperchromatic nuclei; they present a difficult task for the pathologist especially when diagnosis is requested from small curetted pieces of tissue. It is with reference to these cases that Platou said: "It is deplorable that a microscopic examination sometimes permits only of a probable

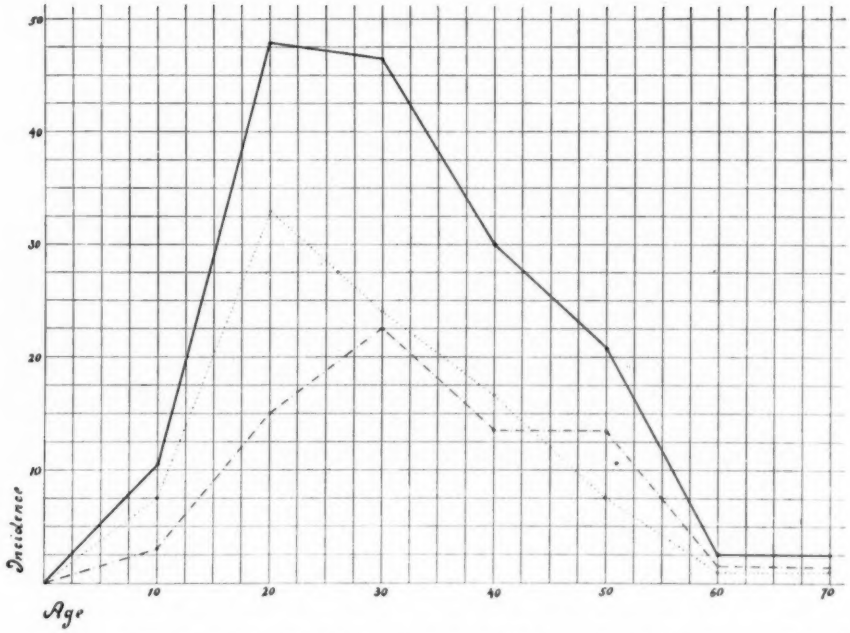


Fig. 92. Curve illustrating the incidence of giant cell tumor in relation to age. Dotted line—females; broken line—males; heavy line—both sexes combined.

diagnosis." Despite this fact it should not be understood that in general the diagnosis of giant cell tumor requires all available data together with an exploratory incision. To the careful observer a complete history and thorough physical examination, supplemented by satisfactory roentgenograms, will suffice for an accurate diagnosis in the majority of cases.

In attempting a diagnosis of giant cell tumor from the clinical findings alone, one should remember that the most frequent error is mistaking it for osteomyelitis; this is at least not less frequent than mistaking it for an osteogenic sarcoma. Because of the location of giant cell tumor in the ends of long bones and also because of its long duration and slow progress, it is the tuberculous osteomyelitis and the tuberculous joint involvement with which one is mostly concerned in such cases. This is especially true when, as it frequently happens, the giant cell tumor is situated about the knee joint. The asymmetric enlargement of only one side of the region of the knee joint and not of the whole area as in tuberculosis, and the absence of the fusiform swelling of the joint, which is frequent in tuberculosis, are strong differentiating points. To depend entirely upon the laboratory specific tests for tuberculosis is not recommended because of their frequent failure as an accurate diagnostic means. In a patient who later died of an osteogenic sarcoma

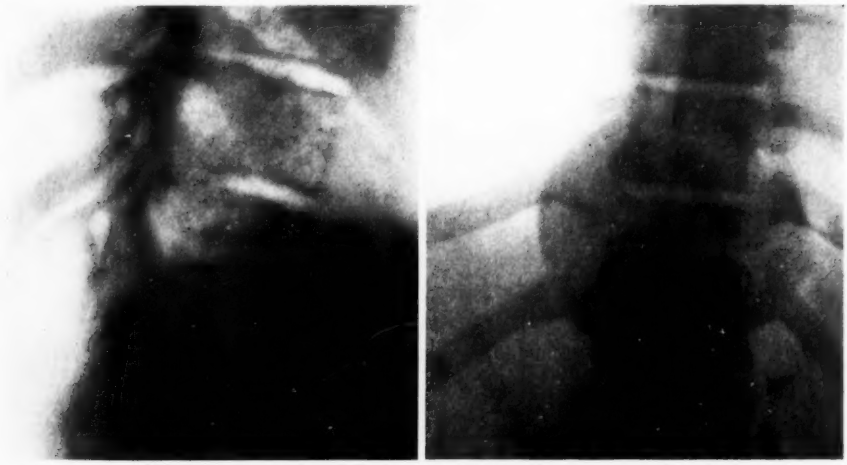


Plate 38. Courtesy of the Ruptured and Crippled Hospital, New York City. Primary sarcomatous tumor of the spine. The tumor arose in the tenth dorsal vertebra which is extensively destroyed while the intervertebral discs remain intact.

without evidence of tuberculous involvement at autopsy, I have seen, following an intradermal tuberculin injection, a positive local, focal, and general reaction. Giant cell tumor of the spine is frequently confounded with tuberculous spondylitis. Nervous disturbances as a result of pressure on the spinal cord and nerve roots are likely to appear earlier in a tumor involvement than in tuberculosis. The pain is of more or less intermittent character in the beginning of the disease and abscesses are absent even in well advanced stages of the tumor. While in Pott's disease the intervertebral discs are destroyed early, in giant cell tumor and also in osteogenic sarcoma and Ewing's sarcoma the destruction of the vertebral body greatly precedes that of the discs (Plate 38). Another sign of giant cell tumor of a vertebra, which is especially significant in the presence of a malignant tumor, is œdema of the soft tissues overlying the involved area of the spine.

The outstanding diagnostic importance of the radiological examination goes without saying. To be of real value and not misleading, the roentgenogram must be irreprehensible. All the important technical points mentioned in the chapter on osteogenic sarcoma are true also here. Repeated radiological examination of skeletal tumors from many angles is indispensable for an accurate diagnosis. The roentgenogram is frequently of more importance than a microscopic examination when a variant of giant cell tumor is dealt with; so for instance in cicatrizing giant cell tumor the histology will be that of a fibrosarcoma but the roentgenogram will be typical of a giant cell tumor.

The radiological appearance of a giant cell tumor is most characteristic (Figs. 93 to 100). It usually casts a bulky spherical shadow showing a multicystic

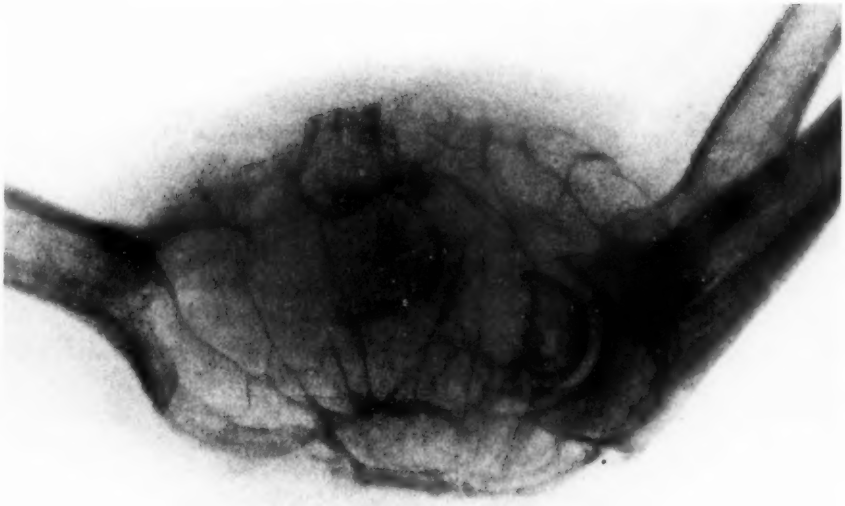


Fig. 93. Courtesy of Memorial Hospital, New York City. Typical roentgenogram of a giant cell tumor.

appearance as a result of the osseous trabeculation in the periphery. The shaft of the bone is absent and it appears as if the cortex is blown out from within the medullary cavity so as to form the thin bone shell, which sharply limits the tumor from the surrounding soft tissues. Beyond the tumor the cortex and the periosteum are entirely unaffected and no periosteal lipping is seen. In the clear roentgenogram an apparent continuation of the bone shell is seen limiting the tumor from the adjointing unaffected medullary cavity; this shadow is an important diagnostic feature and deserves much attention. Not infrequently this borderline shadow is not seen in the roentgenogram because of unsatisfactory technique. To show it requires care in adjusting the position of the tube in relation to the tumor and a rather exacting localizing technique. When the diaphyseal end of the tumor is brought into the field of central rays of the tube it is rarely missed. When in advanced cases of giant cell tumor the bone shell is destroyed, in some areas the roentgenogram will simulate an invasion of the tumor into the soft tissues in spite of the intact periosteum; it should be remembered, therefore, that such a break in the continuity of the bone shell is not a sign against giant cell tumor. When the whole bone shell is destroyed it may be difficult to differentiate it from osteogenic sarcoma but frequently roentgenograms from various angles together with palpation and the clinical history will help in the diagnosis. The fact that the adjoining periosteum and cortex remain unaffected speaks strongly against osteogenic sarcoma. Usually one or another view of the tumor in the roentgeno-

Fig. 94. Courtesy of Memorial Hospital, New York City. Typical giant cell tumor.

gram will show in osteogenic sarcoma the shaft running in the tumor, while in giant cell tumor the shaft is absent. An important differentiation point is also the fact that in osteogenic sarcoma the tumor does not reach the articular cartilage, a layer of spongiosa a millimeter or two in thickness remaining between the tumor and the cartilage, while in giant cell tumor, the tumor is in direct contact with the cartilage. Another condition which requires ruling out in the diagnosis of giant cell tumor is bone cyst. In the majority of the cases this is, however, not difficult. A cyst is intra-osseal and expands the bone only slightly. The cortex of the bone at both poles of the cyst is thinned out so that there is gradual thinning of the cortex from the poles to the middle of the cyst and not an abrupt transition of the normal cortex in a greatly expanded thin trabeculated shell as in giant cell tumor. A cyst is mostly located in the shaft, while a giant cell tumor is in the ends of long bones. The situation of the tumor is not infrequently decisive; if a tumor is in the condyle of the femur or in the tuberosity of the tibia and before breaking through the shaft it extends into the diaphysis, a giant cell tumor is to be considered. It is generally believed that in children a spontaneous fracture as a symptom of onset in a central lesion is characteristic of a cyst and not giant cell tumor. Occasionally a far advanced fibrocystic lesion, when it expands to the epiphyseal and of the bone, may suggest a diagnosis of giant cell tumor. However, in osteitis fibrosa cystica, while the involvement extends far along the shaft, it is not accom-

panied by a corresponding expansion of the bone in transverse diameter, as is seen in advanced giant cell tumor. Osteitis fibrosa is more apt to occur in old than in young people (Figs. 101, 102).

Fig. 95. Courtesy of Memorial Hospital, New York City. Typical giant cell tumor.

When combined with the clinical history, physical findings and radiological features, the data of a pathological examination are of valuable diagnostic importance. The main sources of error in the diagnosis of giant cell tumor from the histology should be mentioned here. It may be safely argued that to one experienced in the pathology of bone tumors the gross anatomy of a giant cell tumor will frequently mean more than the histology, and a diagnosis from the gross specimen will be more apt to be accurate than when the diagnosis is based upon the slides alone. It is well to remember that an occasional tumor, however, can resemble grossly or histologically a giant cell tumor and not be one. The histology is frequently misleading in giant cell tumor; especially so when a frozen section is relied upon, as often happens. The diagnosis of giant cell tumor from the section must not be based upon the presence of giant cells alone; the type of the supporting tumor cell is most important. Although the giant cells are an integral part of the giant cell tumor they are occasionally encountered also in typical osteogenic sarcoma where lime salts are set free by rapid erosion and disintegration of bone. This fact is not realized sufficiently by the average surgeon and pathologist who have only a casual acquaintance with the pathology of bone tumors, and it is a con-

Fig. 96. Case 295. Same case as that in Figure 97. Giant cell tumor in a man 25 years old. This roentgenogram was taken at the time of onset.

stant source of grave errors. Another fact which is not sufficiently realized is that very vascular, so-called telangiectatic osteogenic sarcoma may resemble grossly vascular giant cell tumor. There also one finds giant cells of the epulis type about extravasations. The histology is not infrequently misleading in the variants of giant cell tumor. So, for instance, in the myxomatous variation the histology may suggest malignancy while the clinical findings and radiological features clearly indicate the benign nature of the lesion. In cartilaginous giant cell tumors in a few cases a diagnosis of myeloma has been returned by the pathologist because of the abundance of rounded cells of the stroma. The advanced cicatrizing stage of giant cell tumor may suggest fibrosarcoma, if other data are disregarded. Occasionally several variants may be found in the same tumor. The histology is especially deceiving when the tissues were taken from tumor masses fungating through a former exploratory incision. It is extremely hazardous to base a diagnosis on findings from these granulation tufts. The numerous mitoses here are mostly in the endothelial leucocytes.

The question of indication and contra-indication of biopsies in giant cell tumor is still under discussion. It would seem that the whole question is merely a part of the general problem of the advisability of a biopsy in bone tumors of doubtful nature, since the necessity of exploration indicates that one is not certain whether the tumor is benign or malignant. The difficulties encountered in a diagnosis from the histology in a doubtful giant cell tumor are great and insurmountable to those little initiated in the pathology of bone tumors. As a general rule, when the clinical findings and the roentgenogram are baffling to the clinician, the histology is also distressing to the pathologist. Whatever one may say about the possibility of increase of malignancy in giant cell tumors by an exploratory incision one cannot deny that the dangers of infection with which such explorations are

Fig. 97. Case 295. See Figure 96. This roentgenogram was taken 1 year after the onset. At that time radiation therapy was instituted which led to a shrinkage of the tumor. The patient is earning his living. This is the largest giant cell tumor in the Registry collection. The radiological features are typical of giant cell tumor.

entailed are very great. The spongy vascular tumor and the exposed bone-marrow are very susceptible to infection, and a cellulitis, suppurative osteomyelitis and septicæmia are not rare after such procedures. However, if a biopsy is done the curettage of the tumor should be completed because a following infection will add greatly to the difficulties of radiation. To be decisive a biopsy should include all parts and not only the periphery of the lesion. The radiation therapeutic test is not of such great importance in giant cell tumor as in osteogenic sarcoma

Fig. 98. Case 5. The same case as that in Figures 88 and 89. Giant cell tumor in a boy of 16. The trabeculation in a giant cell tumor of the upper end of the humerus is usually of a more delicate structure, the involved bone appearing honeycombed.

and especially in Ewing's sarcoma however, we have a differentiating point in the fact that pain in giant cell tumor persists after radiation is begun, while in malignant tumors it is usually relieved sooner. In the rare case of an aggressive, rapidly growing tumor which has reached a far advanced stage when first seen, radiation and perhaps an exploratory incision would seem to be better justified than a direct amputation of the limb with only a probable diagnosis of malignancy in one's mind.

Therapy

While in malignant bone tumors the problem of therapy today is to find a way to relieve the sufferings of the patient for a longer period of time, in giant cell tumor the whole crux of the question is as to choice between various methods of treatment, each of which may lead to permanent cure. The history of the therapy of giant cell tumor is remarkable for the continuous change from radical to more conservative methods of treatment. The almost universally employed amputation of some twenty-five years ago was followed by partial resection which later gave way to curettage; and it was curettage that constituted the standard method of therapy in giant cell tumor until very recently. That spontaneous cures of giant cell tumor occur is sufficiently attested; such instances are probably more frequent than is suspected. Toxin treatment has not been applied as frequently as in malignant bone tumors, the adepts in this method of treatment advocating the use of toxins merely as a prophylactic along with radiation after incomplete surgical operations.

Fig. 99. Case 542. Giant cell tumor in a woman of 49. The seat of the tumor in the lower end of the ulna is uncommon, the lower end of the radius being the favored seat of a giant cell tumor about the wrist.

Fig. 100. Case 195. Benign osteogenic tumor. The presence of the shaft of the bone and the fact that the tumor is extracortical speaks against giant cell tumor; the clear cut outline of the tumor speaks against osteogenic sarcoma.

The most frequently employed method of curettage consists of the following procedure: curettage of all removable portions of the tumor followed by swabbing out of the rough curetted area with pure carbolic acid and alcohol, or as some advise, with zinc chloride. The subsequent packing of the cavity with gauze to

prevent hæmorrhage has been given up by some men, who close tightly the operative wound expecting an organization of the blood clot. A promiscuous use of this device in all cases of giant cell tumor should be warned against, since fatal hæmorrhages are known to have occurred. On the other hand if one packs the cavity with gauze, infection is frequent notwithstanding all preventive measures. Whatever the way of dealing with the cavity, the limb should be immobilized to prevent a pathological fracture which will still further complicate the course.

As a prophylactic against a recurrence, radium therapy is to be recommended. Recurrence seems to be frequently unavoidable and is observed in about 20 per cent of all cases treated by a single curettage alone. The cause of recurrence is not known. The histological structure of the tumor gives little explanation of this. The question whether the bone shell is intact or not does not seem to be the decisive factor. That the completeness of curettage has little to do with

Fig. 101. Courtesy of Warren Museum, Boston. Osteitis fibrosa of the tibia in a man of 87. The line of demarcation from the normal bone; the newly formed trabeculæ, the effort of nature to form a buttress behind the bent portion; the intact periosteum and the bowing of the bone are all typical of advanced osteitis fibrosa. Amputation was performed in 1872 by Bigelow.

Plate 39. Case 319. Giant cell tumor in a man 38 years old. Onset in September, 1922. Roentgen-ray irradiation on October 26 and 28, 1922, and on March 13 and 23, 1923. The roentgenograms were taken: October 15, 1922; January 15, 1923; April 18, 1923; May 21, 1924. Function of wrist completely restored.

Fig. 102. Personal observation; not registered. Osteitis fibrosa cystica in a man 68 years old. The lesion was mistaken for a malignant tumor and radiation therapy instituted. Because of an ensuing pathological fracture amputation was done; the histology proved the lesion to be a typical osteitis fibrosa. The wide extent of bone involvement with the relatively small expansion of the bone suggested the diagnosis of osteitis fibrosa from the roentgenogram.

the tendency of the tumor to recur is indicated by the fact that occasionally completely curetted tumors recur while those incompletely curetted go on to a permanent cure. That cauterization is effective only to a relatively small depth is obvious from the chemical coagulative action of the escharotic. In the light of these facts one can never be certain that curettage will not be followed by a recurrence of the tumor. An even greater disadvantage of curettage is infection. This serious complication has occurred in good technical hands and under the best of conditions. The seriousness of infection in a giant cell tumor has been mentioned above. A proven potential danger of repeated curettage of a giant cell tumor is the surgical trauma by itself. I am referring later to the possibility of malignant transformation in rare instances of giant cell tumor after repeated surgical operations and infection.

It has long been known that radiant energy is an active factor as a prophylactic against recurrence of a curetted giant cell tumor. Radiation has for years been used also when the tissue removed during incomplete surgical operations in cases of giant cell tumor seemed to be suspicious. Experience has shown that if radiation is used after curettage no recurrence follows, but the cavity left after curettage does not fill in with new bone and surgery may be the final issue. More disappointing is the experience with radiation therapy of recurrence after curettage. The patient is usually promptly relieved of pain; the infection is checked soon but the growth itself is more refractory to massive radiation. Little bone repair is seen, the scar from the previous operation and even the skin may break down and add considerably to the difficulties of radiation. If curettage has been chosen and done, it should be done repeatedly if recurrences take place, and if necessary, amputation should be considered rather than a change to radiation. Men of wide experience with radiation therapy of giant cell tumor decidedly state that an incomplete surgical operation in giant cell tumor is worse than none.

During recent years a further and most significant step has been taken toward conservative treatment of giant cell tumor; it consists of substituting radiation therapy, either roentgen ray or radium, for surgical operations. It is true that the use of radiation as a primary therapeutic measure in giant cell tumor is not yet widely adopted but the experience accumulated in a few leading medical institutions, especially at the Memorial Hospital in New York, is of such weight that it eliminates any doubt as to the very great value of radiation therapy as a primary therapeutic measure in giant cell tumor, and cases of giant cell tumor cured by radiation are on record. In the reaction of a giant cell tumor to effective radiation therapy one can distinguish two opposite phases. During the first three or four months of radiation therapy the giant cell tumor increases in size, the bone shell expands greatly, becomes thin and finally melts away entirely, the skin becomes reddened, œdematous, inflamed, sore to the touch, and the whole appearance indicates a rapid growth of the tumor. The tumor acquires very suggestive

Plate 40. Courtesy Memorial Hospital, New York City. Giant cell tumor. The roentgenograms were taken (left) before radiation therapy was begun and (right) 3 years and 8 months later.

marks of a malignant sarcoma. This excessive reaction with bone absorption may be avoided by using a less severe radiation dosage. The preliminary reaction of the tumor to radiation is the destructive or negative phase in contrast to the final productive or positive phase of reaction. The latter is characterized by a constant shrinkage of the tumor and by formation of new dense bone. In contrast to the soft and pultaceous consistency of the tumor during the negative phase, it becomes gradually harder in the positive phase of reaction as a result of abundant calcium deposits (Plates 39, 40 and 41). The younger the patient and the more superficial the situation of the tumor the more prompt will be the reaction. In the opinion of those with widest experience, external radiation with roentgen ray or a radium pack is far superior to internal radiation when radium needles are placed into the tumor substance. It is also conceded that the roentgen ray is to be preferred to radium. It is especially dangerous to use unfiltered radium in tumors after curettage; in general, burying unfiltered radium within tumor tissue may lead to bone necrosis, chronic osteitis with abundant sloughs, and infection. The dangers of overradiation should not be underestimated even when external radiation alone is used, when in exceptional cases exposures extending over many months may be followed by radiation changes in the soft parts surrounding the tumor, which may lead to ulceration of the skin, and, in extreme cases, to osteitis with agonizing pain, which may require amputation.

Even if, at the present time, radiation therapy has not as yet become the standard therapy of giant cell tumor there are cases in which only radiation therapy should be considered. This is the case in the giant cell tumor of the spine and in the rare instances of giant cell tumor of the skull. In such cases no complete surgical operation is possible, because of the tendency of the tumor to widely involve the fibrous dense tissue and also of the possibility of a fatal hæmorrhage ensuing after an attempt at surgical removal.

Although the general tendency in recent years has been to substitute conservative surgical procedures and radiation for the radical amputation, the latter, however, cannot be entirely avoided in some rare instances. Occasionally the possibility of a better practical result will decide for amputation rather than curettage. This is sometimes observed when the tumor is situated about the knee, or ankle joint. Infection after biopsy and curettage with suppurative osteomyelitis and cellulitis will make amputation the issue. When the giant cell tumor is exceptionally large the best chance for a cure can be expected from a radical surgical procedure alone since neither curettage nor radiation can cope with the large mass.

In rare instances the histology of the tumor after curettage may appear suspicious enough to call for amputation. Problems in plastic surgery may come up after cure has resulted from curettage in some cases of giant cell tumor of the jaws.

Plate 41. Courtesy Memorial Hospital, New York City. Giant cell tumor with a pathological fracture. Immobilization and radiation therapy instituted. The roentgenogram to to right was taken 22 months after the roentgenogram on the left. The function of the limb is completely restored.

Prognosis

In the preceding chapters I have attempted to stress the importance, in dealing with a giant cell tumor, of a combined evaluation of all data available. This is especially true in relation to prognosis. Only in the light of data accumulated from the clinical history, physical and radiological findings, and pathological examination may a prognosis be given. As in osteogenic sarcoma there are too few cases on record of the giant cell tumor being allowed to follow its natural course without interference by surgery or radiation for definite conclusions as to the natural outcome of such cases to be made. However, there is some evidence on hand to show that when left alone the giant cell tumor may follow one of two ways. Advanced growth of the tumor may lead to fatal hæmorrhage or septicæmia. Occasionally, following a pathologic fracture or without it, such a tumor

Fig. 103. Case 349. See Figure 104. Typical giant cell tumor of the tibia. The photomicrograph made from a section of tissue removed at the first operation (curettage).

may enter a cicatrizing stage in which the peripheral portion of the tumor is being converted into a dry, firm, fibrous tissue. The bony shell may become very thick. In the central portion, islands of active giant cell tumor will persist although some of them may degenerate and be replaced by cysts.

The question of the prognosis of giant cell tumor treated conservatively, by curettage or radiation, forms a subject for ardent discussion, and a voluminous literature has grown up about it which unfortunately is somewhat dogmatic on both sides. On one side many cases are cited of giant cell tumor leading to pulmonary metastases and death. Two sources of error of these authors are revealed by a careful study of these cases. The first is that not in all cases mentioned was the primary lesion a giant cell tumor and the second that not in all fatal cases are metastases proven to have been present. On the other hand the authors believe that giant cell tumor is always a benign lesion lacking the ability to produce metastases. The exceedingly few cases of giant cell tumor in which, after repeated surgical operations, pulmonary metastases and death occurred these authors explain by the fact that due to surgical insult and ill advised therapeutic measures the giant cell tumor became transformed into a malignant bone tumor which,

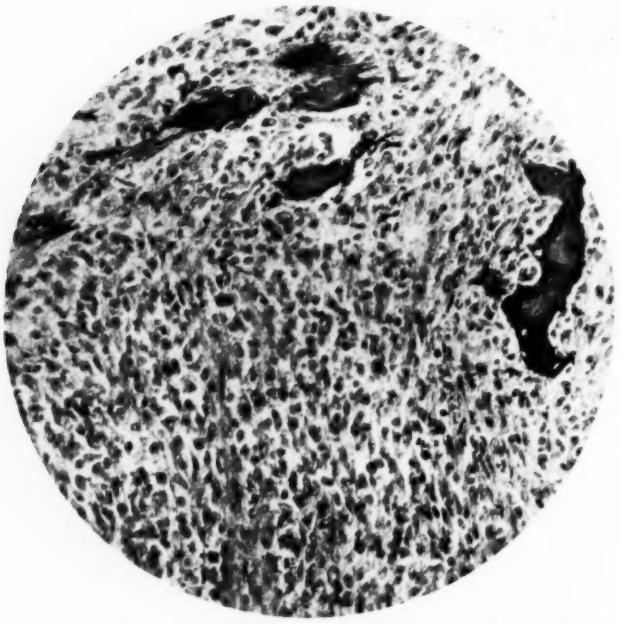


Fig. 104. Case 349. See Figure 103. Structure of the tissue removed from the leg amputated after repeated curettage operations. The histology resembles osteogenic sarcoma. Death with pulmonary metastases.

as a malignant tumor and not as a giant cell tumor, led to pulmonary metastases. It is obvious that such an argument is based solely upon technicalities which do not in the least change the fact that in a tumor diagnosed by experts as giant cell tumor the outcome was death due to pulmonary metastases. That giant cell tumor is essentially a behign lesion is generally conceded. It is benign in the oncological sense of the word although clinically it may offer serious surgical problems. The question as it stands today is whether or not a giant cell tumor is *always* benign. From the evidence on hand this question is to be answered in the negative. Giant cell tumor is essentially a benign condition and there is no case on record in which histologically a typical giant cell tumor gave pulmonary metastases of typical giant cell structure. In the few cases recorded in the literature of death following pulmonary metastases of a giant cell tumor, either both the primary lesion and the pulmonary metastases showed a typical giant cell tumor structure, or the primary lesion was a typical giant cell tumor but the pulmonary metastases were distinctly atypical for giant cell tumor histology. In exceptional cases a giant cell tumor may become a more malignant tumor as a result of repeated irritation—curettage and infection (Figs. 103 and 104).

The main rule that giant cell tumor is essentially a benign lesion is strongly supported by the fact that these exceptional cases are exceedingly rare; inasmuch as the possibility of forcing tumor cells into the blood channels during curettage is very great in so vascular a lesion. Cases have been seen of recovery following resection of a giant cell tumor which on dissection showed the presence of tumor cells growing in a vein. To produce metastases it is not sufficient to have tumor cells in the blood stream; the cells must be sufficiently viable to be able to settle down and to multiply at their new place. But when, after repeated irritations, the tumor cells become more resistant and viable they may acquire malignant features. This possibility that a giant cell tumor may become transformed into a malignant new growth is additional proof that at least in some instances giant cell tumors are true tumors and do not represent merely a repair reaction to inflammation.

The main factor in the clinical course of giant cell tumor requiring one to be on guard in the prognosis is rapid growth of the tumor. The typical giant cell tumor is of long duration and slow course. Rapidity of growth is a sign of aggressiveness of the tumor. Another reliable sign of aggressiveness is the destruction of a large portion of the bone shell of the tumor. The presence of the bone shell is important not because its absence is proof that the tumor has invaded the surrounding soft tissues but because destruction of the bone shell is in a moderately advanced tumor an indication of the aggressiveness of the new growth. A giant cell tumor reaching a very large size is very apt to recur after curettage since the size of the tumor excludes the possibility of a complete removal of the tumor tissue. With each recurrence the prognosis becomes less favorable. Care should be exercised in the arrival at a prognosis based upon a recurrence, because with each recurrence the growth is apt to become more anaplastic and malignant.

In relation to a prognosis the findings of a pathological examination are of outstanding significance. The giant cells of epulis type when present in excess are a true indication of the benignity of the lesion. The typical giant cell tumor where the giant cells form the bulk of the tumor is of very slow growth, not aggressive, and easily eradicated even by incomplete curettage. On the other hand with the disappearance of the giant cells and with an increase in the number of the spindle cells of the stroma the aggressiveness of the tumor increases. Viewed largely, the type of cells of the stroma is of greater importance than the giant cells. It is by the various numbers, sizes and shapes of the stroma cells that varieties of giant cell tumors are distinguished. Increase in the hyperchromatism of the stroma cell calls forth a warning as to the likelihood that a recurrence will take place after an incomplete surgical operation. A recurrence can be also expected when the stroma cells have become abundant and rounded. However, pleomorphism and cellularity in the central portion of a giant cell tumor

are not of such unfavorable significance for the prognosis as their presence in the peripheric portion of the tumor, where they notoriously mark aggressiveness. A guarded prognosis is to be given also in the case of the very vascular, so-called telangiectatic, giant cell tumor which as a rule is more aggressive and recurs more frequently than other types.